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POCKET HANDBOOK OF SMALL ANIMAL MEDICINE

SECOND EDITION

Kit Sturgess

Pocket Handbook of Small Animal Medicine

Concise, problem-oriented, and accessibly designed, the second edition of the *Pocket Handbook of Small Animal Medicine* focuses on the essentials of medicine and surgery of the dog and cat. Packed with bullet points, flowcharts, and tables for quick reference, the book helps start the process of case management and resolution. It includes tips and hints on interpretation of physical signs, diagnostic tests, and management of common presenting conditions. Full colour algorithms assist decision-making, specifically in clinical presentations and critical care.

- Part 1 covers an overview of basic approaches.
- Parts 2 and 5, 'Clinical Presentations' and 'Critical Care', present a series of algorithms intended to guide the reader from presentation to diagnosis, or to outline emergency management.
- Part 3 forms the core of the book, covering – in brief – the examination, diagnosis, and treatment of the diseases (including a new section on exotic infections) and disorders of each body system, as well as multisystemic disorders.
- Part 4 covers anaesthetics, analgesia, and surgery.

This new edition is completely updated and contains important new sections on client communication and professional conduct, mental health and wellbeing, therapeutics, and common procedures.

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Pocket Handbook of Small Animal Medicine

Second Edition

Kit Sturgess
MA, VetMB, PhD, CertVR, DSAM, CertVC, FRCVS, RCVS
Recognized Specialist in Small Animal Medicine, Advanced Practitioner in Veterinary Cardiology

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DEDICATION

This second edition is dedicated to my wife, Avery, and my children, William and Matthew, for the family time they gave up allowing me to write this text; my parents' memory for their endless encouragement and support; and the veterinary teams whose support, wisdom, and insight are a constant inspiration and without whom I would not have gained my knowledge and experience.

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PREFACE

Updating should be easy – it has been uplifting to write this second edition as it has served to remind me how much the profession has grown in the ten years since the first edition, how much I have learnt and the greater emphasis on evidence-based medicine. I hope that I have retained the right balance for this edition with extra sections in Part 1 on techniques, therapeutics, non-clinical skills, and wellbeing that are essential to successfully managing our patients.

The Pocket Handbook of Small Animal Medicine remains a 'pick-up-and-get-started' text for quick and easy reference. It aims to offer concise subject reviews and rapid interpretation and problem-solving of key clinical signs. Part 1 still covers an overview of basic approaches, general health concerns, while Parts 2 and 5, 'Clinical Presentations' and 'Critical Care', present a series of algorithms intended to guide the reader from presentation to diagnosis, or to outline emergency management. Part 3 forms the core of the book, covering – in brief – the examination, diagnosis, and treatment of the diseases (including a new section on exotic infections) and disorders of each body system, as well as multisystemic disorders, while Part 4 covers anaesthetics, analgesia, and surgery. For more in-depth discussion and treatments, a short Further Reading list is included.

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ABOUT THE AUTHOR

Dr Kit Sturgess has been in clinical practice for nearly 40 years in primary care, university, and private referral clinics He was awarded his PhD looking at the mucosal immune response to feline immunodeficiency virus (FIV). Kit is a Specialist in Small Animal Medicine and an Advanced Practitioner in Veterinary Cardiology. He was awarded his Fellowship of the Royal College of Veterinary Surgeons for Meritorious Contributions to Clinical Practice and the BSAVA Woodrow Award for contributions to small animal practice in 2022. Besides clinical work Kit spends his time writing and lecturing and has had a wide variety of voluntary roles including Chair of Trustees of Cats Protection and the Wildheart Trust, Editor-in-Chief of *Veterinary Evidence*, Chair of BSAVA Education Committee, and Council member and Treasurer of the RCVS. He maintains a keen interest in many areas of internal medicine, particularly endocrine, cardiorespiratory, and gastrointestinal disease, and has authored numerous articles and two textbooks as well as examining and presenting lectures and research abstracts at conferences worldwide.

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1

BASIC APPROACHES

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1.1 GENERAL HEALTH

1.1.1 *Dog breeds* 1.1.2 *Cat breeds* 1.1.3 *Congenital and hereditary disease* 1.1.4 *Genetic defects* 1.1.5 *Genetic screening* 1.1.6 *Vaccinology* 1.1.7 *Parasite control* 1.1.8 *Feeding healthy pets* 1.1.9 *Influence of life stage and lifestyle* 1.1.10 *Feeding the hospitalised patient; tube-feeding techniques*

INTRODUCTION

- While lifestyle and care clearly affect pet health, disease risk is also affected by genetic factors (1.1.3–1.1.5).
- The ultimate goal of medicine is to prevent patients becoming sick. Vaccination (1.1.6), parasite control (1.1.7), and appropriate nutrition (1.1.8) reduce this risk whilst health screening in older patients serves to spot occult disease allowing earlier intervention with the expectation (although generally poorly evidenced) of prolonging life and delaying the onset of clinical disease.
- In the UK, microchipping is now compulsory in dogs and in some nations in cats – likely to become UK wide. It is the role of the veterinary team to ensure clients are aware of their legal obligations but not to enforce them.
    - Reminding clients to update details on the microchip database if they move or there is a change in ownership is good practice.

BREEDS, BREEDING, AND DISEASE

1.1.1 DOG BREEDS

- Dogs have been bred with an incredible variety of body shapes and sizes, with over 220 breeds divided into ten groups by the Kennel Club.
- The general trend in the UK over the recent past has favoured smaller dog breeds.
- There are increasing concerns about the health and welfare of some breed groups, in particular the brachycephalics with extreme conformations. There has also been an explosion of first cross 'breeds' – labradoodles, cockapoos, jugs. Unfortunately, evidence suggests that 'hybrid vigour' has not resulted in fewer health problems.
    - Group 1: sheepdogs and cattle dogs.
    - Group 2: pinschers and schnauzers, Molossians, Swiss cattle dogs.
    - Group 3: terriers.
    - Group 4: dachshunds.
    - Group 5: spitz and primitive Nordic breeds, English spitz, Asian and related spitzes, primitive types, primitive hunting dogs.
    - Group 6: scent hounds, leash hounds, related breeds.
    - Group 7: continental pointers, Irish pointers and setters.
    - Group 8: flushing dogs, retrievers, water dogs.
    - Group 9: companion and toy dogs (12 sections, including poodles, bichon, Tibetan, Dalmatian, Japanese chin).
    - Group 10: sight hounds and related breeds.
- The PDSA Paw Report (2023) estimates the UK dog population at 11 million of which 6% had travelled abroad in the past year and 6% were acquired from abroad, making 8% of the UK dog population bred outside the UK.
- In the UK, around 70% of dogs are specific breeds with French bulldogs having overtaken the Labrador retriever as the most popular breed.
- Cockapoos, cocker spaniels, dachshunds, Staffordshire bull terriers, Jack Russell terriers, border collies, and Chihuahuas being amongst the most common breeds.
- See www.thekennelclub.org; www.pdsa.org.uk for further information.

1.1.2 CAT BREEDS

- There are around 50 registered breeds of domestic cat, grouped in the UK as follows by the Governing Council of the Cat Fancy (GCCF).
  - Persian.
  - Semi-longhair: Birman, Maine coon, ragdoll, Somali, Norwegian forest cat.
  - British shorthair, including Manx and Selkirk rex.
  - Foreign: Abyssinian, Bengal, rex, Russian, Korat, sphynx.
  - Burmese.
  - Oriental: including Havana.
  - Siamese: including Balinese.
- The Cats Protection CATS Report (2023) estimates the UK cat population at 11 million. With 67% of households having a single cat. Online sales accounted for three-quarters of cats bought. Around a quarter of owned cats are specified breeds with British shorthair (BSH) being the most popular by far.
- BSH, Siamese, Bengal, Persian, Maine coon, ragdoll, and Scottish fold account for 65% of pedigrees.
- See www.cats.org.uk and www.gccfcats.org for further information.

1.1.3 CONGENITAL AND HEREDITARY DISEASE

- Animals born with any defect have ***congenital disease***, which does not have to be heritable.
- ***Heritable disease*** may not be seen until later in life, e.g. mitral valve disease in Cavalier King Charles spaniels.
  - The same phenotype, e.g. cleft palate, can occur both as a heritable and non-heritable congenital defect in cats.
  - If a litter is born with or develops a defect, the hallmark of heritable disease is that only a proportion of the litter is affected by an identical defect.
- If all or most of a litter is affected and the defects are different, this indicates an insult *in utero*, not genetic defects.
- *De novo* genetic defects can occur in an individual, mostly these are silent but in a few a new disease occurs.

1.1.4 GENETIC DEFECTS

- Genetic defects can be:
  - Breed related: where the fact of being of that breed predisposes the animal to a disease (e.g. brachycephalic airway syndrome in bulldogs) and breeding out a defect would change the breed considerably.
  - Breed defects: defect is widespread in multiple lines within the breed, e.g. flat chests in Burmese, hip dysplasia in retrievers.
  - A defect reported in a breed: many defects that are reported as breed defects have only appeared in a few individuals or specific lines within the breed. However, the true prevalence of the genetic tendency within the breed may be masked by individuals' lack of expression, e.g. craniofacial malformation (USA Burmese; not UK).

1.1.5 GENETIC SCREENING

- Tests are available for an increasing number of diseases, such as polycystic kidney disease in Persians, or von Willebrand disease.
  - The limitations of the particular test should be recognised.
  - Some genetic tests actually identify the defect (most reliable) whereas others identify genotypes that are associated with the disease and are less reliable.
    - As tests identify a specific defect, the individual can still have that genetic disease but associated with a different defect, e.g. there are hundreds of genetic defects identified as causing hypertrophic cardiomyopathy in

people. Veterinary medicine lags far behind the human field in identifying genetic defects linked to specific diseases.
    - Some tests identify genetic traits such as dog leukocyte antigen (DLA) Class II haplotypes, associated with canine diabetes.
- Care should be taken when using a test to check that it has been appropriately validated for that disease in that breed. Careful explanation of the impact of a 'positive' genetic test should be given to the client as it does not necessarily mean their pet will go on to develop that condition.

PREVENTIVE MEDICINE

1.1.6 VACCINOLOGY

Approximately 70% of cats and 80% of dogs in the UK are up to date with their vaccinations. WSAVA (2024) has published guidelines for vaccination of dogs and cats.

- ***Principles***: vaccination primes the immune response. If wildtype infection is encountered, a rapid anamnestic response occurs, resulting in a quicker protective immune response that occurs before the infectious agent has multiplied to a level which would cause clinical signs.
    - Vaccines for allergic and neoplastic disease are not preventative but are aimed at modifying the immune response.
- Vaccination is not 100% safe or effective. The immunogenicity of different infectious agents varies considerably; canine parvovirus is far more immunogenic than leptospires, resulting in stronger and longer-lasting immunity following vaccination.
    - Severe vaccine reactions are rare, although long-term risks associated with vaccination are not fully understood but do exist, e.g. vaccine-associated sarcoma in cats.
    - Vaccination has been loosely associated with immune-mediated diseases but most of the data do not support cause and effect.
    - Vaccine failure can be due to:
        - Animal factors, e.g. already incubating disease or strong maternally derived immunity.
        - Disease factors, e.g. new strains or weight of infection.
        - Vaccine factors, e.g. inappropriate strain inclusion, inappropriate storage or delivery.
- ***Vaccination schedules*** vary with vaccine manufacturer.

The aim of puppy/kitten courses is to achieve early vaccination to allow socialisation.

    - The primary course is generally two vaccines 3–4 weeks apart with the second vaccination at 10–12 weeks old.
        - Modified live vaccination of very young puppies and kittens risks causing the primary disease.
    - Not all puppies and kittens respond to the primary course (e.g. 65% to distemper). A first booster at 15 months is vital to cover all individuals.
        - Note that the duration of immunity for distemper/parvovirus vaccination depends on the manufacturer but will be a minimum of 2–3 years.
- ***Vaccine types***: modified live virus (replication attenuated); killed adjuvanted; genetically engineered, containing part of the agent (adjuvanted or inserted in a replication-deficient carrier).
- Choice of vaccine type (see Table 1.1) depends on local disease prevalence and patient factors, e.g. FIV status.
- ***Serological testing***: prior to vaccination for CDV, CAV, and CPV is popular with some owners to try and ensure vaccines are only given when necessary. Protective antibody

Table 1.1 Vaccines currently available for use in cats and dogs

Dogs	Cats
*Distemper	*Feline Calicivirus
*Hepatitis (CAV-2)	*Feline herpes virus 1
*§Leptospira	*Parvovirus
*Parvovirus	*Rabies
*Rabies	Bordetella bronchiseptica
Bordetella bronchiseptica	Chlamydia
Coronavirus	FeLV
Parainfluenza	FIP (not worldwide)
Leishmania (not worldwide)	FIV (not worldwide)

* Core vaccines

§ All vaccines protect against *L. interrogans* serogroup canicola and *L. interrogans* serogroup icterohaemorrhagiae. In the UK, some vaccines also contain *L. interrogans* serogroup Australis and *L. kirschneri* serogroup Grippotyphosa

titres have been published but false negative (assumed due to strong cell-mediated immunity) and false positive results will occur.

1.1.7 PARASITE CONTROL

- Virtually all puppies and kittens will be born with, or acquire during suckling, a parasite load. Low-level parasitism is also common in adult dogs and cats, though rarely of great clinical significance, it does pose a potential public health risk.
- It is therefore important to develop a clear, practice-based parasite control policy for puppies and kittens, adult dogs and cats, and pregnant bitches and queens.
- The choice of product and frequency of worming will depend on local prevalence (Figure 1.1) of specific parasites, and on the lifestyle of the patient. The impact of worming on the environment also needs to be considered.

Table 1.2 shows major groups of parasiticides available in the UK, their route(s) of administration, and spectrum of activity.

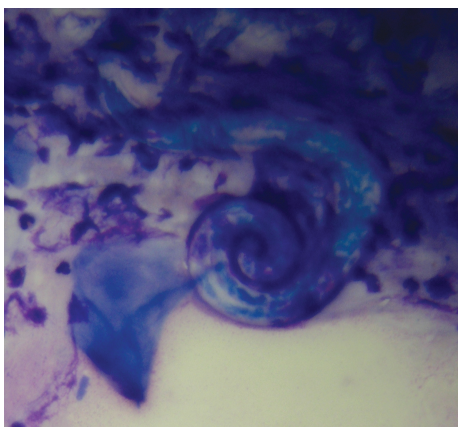


Figure 1.1 Lungworm larva seen on bronchoalveolar lavage. There are several parasitic nematodes, including Oslerus osleri in dogs and Aelurostrongylus abstrusus and Capillaria aerophilia in cats, that can cause pulmonary disease.

Table 1.2 Principal parasiticides for use in cats and dogs by mode of action and active ingredient illustrating their spectrum of activity

			Ectoparasites							Endoparasites												
Mode of Action	**Active ingredient**	**Route**	Fleas	Flies	Ticks	Demodex	Sarcoptes	Otodectes	Lice	Taenia spp.	Dipylidium	Echinococcus	Giardia	Angiostrongylus	Toxocara	Toxocara larvae	Uncinaria	Ancylostoma	Trichuris	Lungworm	Dirofilaria	**Host species**
Acetyl choline blockade	Piperazine*[1]	Oral													•		•					dog/cat
Bind to tubulin disrupting cell function	Fenbendazole	Oral								•	•	•	•	•	•	•	•	•	•	•		dog/cat
	Flubendazole & Mebendazole	Oral								Likely similar activity to fenbendazole												Large animals
Cestocide - interferes oxidative phosphorylation	Dichlorophen	Oral								•	•	•										dog/cat
Cestocide increases cell membrane permeability	Praziquantel	Topical								•	•	•										dog/cat
GABA 'ligand-gated chloride channel blocker'*[2]	Afoxolaner	Oral	•		•	•	•	•	•													dog
	Esafoxolaner	Topical	•		•	•	•	•	•													cat
	Fipronil	Topical	•		•			•	•													dog/cat
	Fluralaner	Oral/topical	•		•	•	•	•	•													dog/cat
	Lotilaner	Oral	•		•	•	•	•	•													dog/cat
	Pyriprole	Topical	•		•																	dog
	Sarolaner	Oral/topical	•		•	•	•	•	•													dog/cat
Inhibition of chitin synthetase	Lufenuron*[3]	Oral/injection	•																			dog/cat
Inhibits fumarate reductase system	Tiabendazole	Ear drop						•														dog/cat

Interferes with GABA and glutamate-gated channels*4	Eprinomectin	Topical/oral/injectable	•		•		•	•	•						•		•			•	•	cat
	Ivermectin*5	Topical				•	•	•	•													dog/cat
	Milbemycin	Oral								•	•	•	•		•			•	•	•	•	dog/cat
	Moxidectin	Topical	•			•	•	•	•						•		•	•	•	•	•	dog/cat
	Selamectin	Topical					•	•	•					•	•	•	•	•	•	•	•	dog/cat
Juvenile hormone analogue inhibits larval development	Methoprene	Topical	•																			dog/cat
	Pyriproxyfen	Spray	•																			dog/cat
Nicotinic acetylcholine receptor agonist	Pyrantel/oxantel	Oral/topical													•		•	•				dog/cat
	Dinetofuran	Topical	•	•	•																	dog
	Spinosad	Oral	•		•																	dog/cat
Octopamine receptor agonist	Amitraz	Topical				•	•															dog
Post synaptic nicotinic receptor binder	Nitenpyram	Oral	•																			dog/cat
	Imidaclopramid	Topical	•		•																	dog/cat
Sodium 'open channel blocker'*6	Deltamethrin	Topical	•	•	•																	dog
	Permethrin	Topical/spray	•		•	•																dog
Stimulates presynaptic secretin receptors	Emodepside	Topical								•	•	•			•	•		•				cat
Voltage dependent sodium channels	Indoxacarb	Topical	•																			dog/cat

*1 Antagonist reaction with pyrantel; toxic to large cats
*2 Isooxazoline parasiticides have similar spectrum of activity but different licensed use
*3 Insect growth regulator
*4 Avermectins parasiticides have similar spectrum of activity but different licensed use; seizures in Collies & related breeds
*5 Only topical licensed in UK for cats and dogs
*6 Toxic to cats

NUTRITION

1.1.8 FEEDING HEALTHY PETS

- Energy requirements depend on life stage, lifestyle, presence of chronic disease, neutering, and reproductive status (females).
- Breed-specific diets have been designed to meet:
  - Anatomic need, e.g. jaw shape in Persian cats.
  - General requirements of a breed group associated with their body size, body shape, appetite, or temperament.
  - Theoretical nutritional strategies to reduce the incidence/severity of diseases common in that breed.
  - Individualised diets based on owner questionnaire responses.
  - Evidence of benefit of breed-specific and individualised diets is theoretical.

TARGET ENERGY REQUIREMENTS

- Metabolisable energy (expressed per 100 g of dry matter for comparison) is used to digest, absorb, and utilise food (heat increment) and is a proportion of the gross energy in a food (Figure 1.2).
- The remainder is available for growth, lactation, reproduction, physical performance (net energy for production) and repair, basic activity, and thermoregulation (net energy for maintenance). Excess energy is then stored as fat.
- ***Resting energy requirement*** (RER) is the basic energy expended at rest:
  - RER (kcal/day) = 70 × (bwt in kg)$^{0.75}$
  - ≈ 30 × (bwt in kg) + 70 (for animals from 2–48 kg).
- ***Maintenance energy requirement*** (MER) is the amount of energy required by a typical adult pet cat/dog (Table 1.3). A basic MER is expressed by the following equation, but it should be further modified according to life stage (Table 1.4) and lifestyle. MER (kcal/day) = 1.2 × 1.4 × RER (neutered dog) 1.4–1.6 × RER (intact) = 55 × (bwt in kg) (neutered cat).

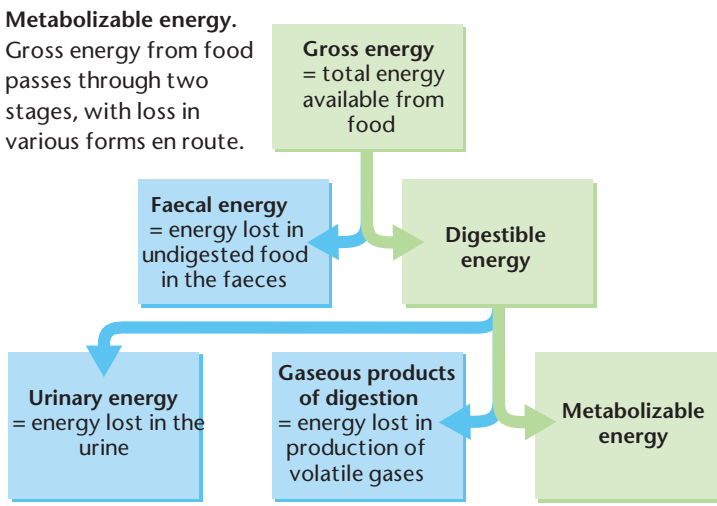

Metabolizable energy.
Gross energy from food passes through two stages, with loss in various forms en route.
Gross energy = total energy available from food
Faecal energy = energy lost in undigested food in the faeces
Digestible energy
Urinary energy = energy lost in the urine
Gaseous products of digestion = energy lost in production of volatile gases
Metabolizable energy

Figure 1.2 Relationship between gross and metabolisable energy.

Table 1.3 Quick guide to maintenance energy requirements by lean body weight in dogs							
Dog Weight (kg)	5	10	15	20	25	30	40
MER (kcal)	320	530	720	900	1060	1220	1500

Table 1.4 Daily energy requirements of dogs and cats at different life stages		
Daily energy requirements (kcal)		
Life stage/lifestyle	**Dog** (× MER§)	**Cat** (kcal/kg bwt)*
<3 months (growth)	2	200
3–6 months (growth)	1.5	130
6–12 months	1.2	55–80
Inactive adult	0.75	55
Active adult	1	60–70
Working 1 hour/day	1.1	Not applicable
Working full day (mild–moderate work)	1.5–2	Not applicable
Gestation (<42 days)	1–1.1	80–90
Gestation (>42 days)	1.1–1.3	90–105
Peak lactation (3–6 weeks)	*Ad lib* feeding or 1 + (0.25 × no. of puppies)	*Ad lib* feeding or 80 × (1 + 0.25 × no. of kittens)
Cold weather (pet outside)	1.25–1.75	No data

§ use MER calculation based on neuter status * – assume neutered by 6 months

Typically, gross energy in wet foods = 1 kcal/g and dry foods = 3.5 kcal/g

Daily energy requirement (DER) also includes requirement for reproduction, growth, and work.

1.1.9 INFLUENCE OF LIFE STAGE AND LIFESTYLE

- Growth
  - Pre-weaning: ideally dam's milk, if not then fostering. Hand rearing requires proprietary milk substitute.
  - Post-weaning: puppies expected to reach 50% and kittens 75% of adult weight by 6 months. Maximal growth does not mean optimal growth.
- ***Puppies*** require energy-dense, highly digestible food. The amount and balance of vitamins/minerals, particularly calcium and phosphorus, are important especially for giant breeds.
- ***Kittens*** are best fed *ad libitum*. Energy requirements per kg peak at 10 weeks, but are lower than for puppies, as the percentage increases over birth weight are less.
- ***Maintenance*** has few requirements outside of a balanced diet.
  - Cats and small dogs reach adult requirements at 12 months, medium dogs 15–18 months and giant breeds 18–24 months.
- ***Gestation*** requires increased feeding as follows:
  - For dogs in fifth–ninth weeks (when most foetal weight gain occurs), increase ration by 15% per week, with small meals of higher density foods.
  - For cats feed *ad lib*: if portion-fed, increase by 4–5% per week.

- ***Lactation*** makes a huge nutritional demand, peaking at 4 weeks postpartum. *Ad libitum* or small meals of highly palatable, highly digestible food required for bitches, *ad libitum* feeding of queens is recommended.
  - Additional vitamins/minerals are not necessary if diet is balanced as total intake increased due to increased amount eaten.
- ***Activity***: a 5 km run increases a dog's requirements by only 10%! Generally, working dogs need a highly palatable, energy-dense (fat), highly digestible, and nutritionally balanced diet.
- ***Old age*** requirements are poorly defined. Intestinal function declines from 8 years of age. It is generally felt that lower nutritional density is required, as energy demands decrease due to reduced levels of activity and lower lean body mass. Vitamin and antioxidant requirement may be increased. Palatability is important as appetite can be reduced. Increase feeding frequency.

1.1.10 FEEDING THE HOSPITALISED PATIENT

- Nutrition needs to be addressed if the patient is severely inappetent/anorexic for >3 days.
- If food manipulation or handfeeding fails, then consider appetite stimulants (see Table 1.5) or tube feeding.

TUBE-FEEDING TECHNIQUES

- ***Naso-oesophageal tube***: can be fitted in a conscious or lightly sedated patient. Inappropriate if vomiting, oesophageal disease, unconscious patient.
  - Tube measured to ninth rib (cat); to seventh intercostal space (dog).
  - Nostril desensitised and tube (6–16 Fr) advanced in a ventral and medial direction with the neck flexed to the premeasured point.
  - Check tube position by connecting a syringe and sucking back to create negative pressure or radiograph. Flushing with sterile water/saline to elicit a cough if intratracheal placement unreliable.
  - Secure tube with sutured or glued tape wings.
  - Good for short-term feeding (max. 5–7 days).
- ***Oesophagostomy tube***: a small incision is made in the oesophagus distal to pharynx under light general anaesthesia and 14–18 Fr feeding tube inserted
  - Position and contraindications as for naso-oesophageal tube.
  - Take care regarding wound infection: there is a low risk of oesophageal stricture.
  - Use for short- to medium-term feeding.

Table 1.5 Dose rates for appetite stimulants used in cats and dogs

Appetite stimulant	Dose rate
Diazepam (cat/dog)	0.1–0.2 mg/kg po or i/v q12–24 hr
Oxazepam (cat)	0.2–0.4 mg/kg po q24 hr
Cyproheptadine (cat)	2 mg/cat po q24 hr
Mirtazapine	1.875 mg/cat po q48 hr 0.5 mg/kg po q24 hr (dog)
Capromorelin*	1–3 mg/kg po q24 hr (cat) 3 mg/kg po q24 hr (dog)

Note: Corticosteroids, anabolic steroids and megoestrol acetate are not first-choice drugs due to their potential side-effects

* not available UK (2023)

- ***Gastrostomy tube***: per-endoscopic gastrostomy (PEG), blind or surgical, 16–20 Fr tube with a suitable mushroom tip. PEG is quicker and much less traumatic than surgery.
  - Risks:
    - Inappropriate if vomiting, unacceptable anaesthetic risk.
    - Risk of leakage around the tube and peritonitis, necrosis of the stomach wall (tube pulled too tight), splenic laceration, interference with gastric emptying.
    - Risks highest with blind and lowest with surgical placement.
  - For long-term feeding (months to years).
- ***Other***: jejunostomy tube feeding and total parenteral nutrition are specialised techniques used in vomiting and unconscious patients.

1.2 HISTORY TAKING AND PHYSICAL EXAMINATION

1.2.1 *Consultation as a problem-solving exercise* 1.2.2 *Consultation as a collaboration with the owner* 1.2.3 *Open and closed questions* 1.2.4 *General background questions* 1.2.5 *Presentation-related questions* 1.2.6 *Examination basics* 1.2.7 *Normal parameters*

GENERAL APPROACH

1.2.1 CONSULTATION AS A PROBLEM-SOLVING EXERCISE

- History taking and physical examination are key to achieving a diagnosis and treating cases appropriately.
- A thorough knowledge of normal variation is important in the interpretation of findings.
- Consultations should be conducted using a problem-solving approach, as this:
  - Helps to apply vast amounts of factual information from a number of different sources to an individual case.
  - Ensures the approach is logical and thorough, allowing important facets of the case to be appreciated, and reducing the risk of missing crucial information.
  - Provides accurate and complete medical records for effective communication between colleagues, thus aiding case management, which is particularly important in a multiperson/multicentre practice.
  - Provides accurate and complete medicolegal records.
  - Identifies critical patient problems.
  - Records less-critical problems which may be important later in patient management or may be of greater significance than originally apparent.
  - Encourages clear decision making.
  - Information gathering should be a balance between open and closed questions (1.2.3), active listening, paraphrasing, repetition and internal summarising.

1.2.2 CONSULTATION AS A COLLABORATION WITH THE OWNER

PLANNING AND MANAGEMENT

- Construct a differential list.
- Explain why each major differential (keep to a maximum of 3–4) is relevant; use visual aids if helpful.
- Relate differentials to owner concerns and check understanding:
  - Explain benefits/drawbacks for each management strategy, include risks, impact on patient, and costs; solicit owner's thoughts.
  - Offer choice of approach, encourage owner to express preference.

- Negotiate an approach that is appropriate, practical, effective, and which the owner understands.
- Check agreement on the plan of action and expected outcomes.

SUMMARY AND CLOSURE

- Summarise and clarify plan of action.
- Make sure the plan is delivered in easily understood language.
- Provide a safety net: a plan B if the response is not as expected.
- Address any outstanding concerns which the owner may have expressed.
- Agree date of follow-up appointment or further action.

CLINICAL HISTORY

1.2.3 OPEN AND CLOSED QUESTIONS

- Owner information is often undervalued. Fitting a full clinical history into a time-restricted consultation is difficult and is often overlooked in the desire to begin the physical examination.
- It is important to address the patient's problems not only as you identify them, but also as they are perceived by the owner.
- When asking an owner questions, where possible they should initially be phrased so as not to imply the answer that is expected.
  - Closed questions invite a yes/no answer – quicker in obtaining a 'history' but may miss vital information.
  - Open questions allow a range of responses, as well as triggering other unasked but potentially important history.
  - Example 1:
    - 'Does Rover have diarrhoea?' (closed) is answered 'No',
    - *but*
    - 'What is Rover's poo like?' (open) receives the answer 'covered in slime with a lot of straining'.
    - 'Is Henry vomiting?' (closed) is answered 'Yes', not understanding the exact medical terminology, when Henry is in fact regurgitating.
  - Example 2:
    - 'Is Fluffy drinking more?' tends to invite the answer 'Yes', partly because the question implies that the owner should know, and partly because it seems that the vet wants this answer to aid diagnosis.
    - 'Have you noticed a change in the amount Fluffy is drinking?' allows the owner more easily to answer that they have not noticed any change – either because they have not looked, or there has been no change.
  - Closed questions are often more appropriate later on in the history when specific facts are being sought.

1.2.4 GENERAL BACKGROUND QUESTIONS

Practice records should not be the sole source of the following information.

- Age, breed, sex.
- Time in owner's possession.
- Travel history.
- Past history from owner or records.
- Worming, vaccination, flea control: this may not be on the medical record as products may have been obtained from other sources, vaccination clinics, and so on.
- Tick history.
- Environment: access to outside (cats), where and how much exercise (dogs).
- Diet – current and recent previous.

- Other pets: are they well?
- Neutering status, or stage in reproductive cycle.

1.2.5 PRESENTATION-RELATED QUESTIONS

- Why has the pet been presented (now)?
- General demeanour.
- Appetite and weight.
- Thirst/urination.
- Defecation, vomiting, diarrhoea.
- Coughing, sneezing, respiratory effort.
- Lameness and pain.
- Lumps, bumps, and skin changes.
- Sleeping and mentation.
- For additional history elements related to specific presentations see Section 2; and related to specific body systems, see Section 3.

PHYSICAL EXAMINATION

1.2.6 EXAMINATION BASICS

- Time should be taken to complete the physical examination although this may not be possible in all patients, for a variety of reasons.
- If parts of the physical examination cannot be completed, e.g. auscultation of the thorax of a purring cat, this should be recorded and the owner should be informed.
- Maximum information will be gained if both the patient and the owner are calm and cooperative.
- Treat seriously any owner accounts of previous issues with physical examination.
- Ask the owner if their pet will react better if the owner holds it or whether you should ask another member of the practice to help.
- Consider examining dogs on the floor: some dogs will be more relaxed there than on a table.
- If you think that you need to use a muzzle, ask the owner if this is acceptable, and about any previous response to muzzling.
- Elements of the physical examination related to specific body systems are presented in the relevant sections.

1.2.7 NORMAL PARAMETERS

HEART AND RESPIRATORY RATES

- Reference ranges for heart and respiratory rates are shown in Tables 1.6a and 1.6b. While absolute heart rate is important, it should also be appropriate for the situation.
    - For example, normal dogs asleep can have heart rates less than 30/min and pauses of 4–5 seconds.
    - A heart rate of 60/min in the consulting room in a highly stressed Yorkie is unexpectedly low but may be expected in a laid-back Labrador.
- The heart rhythm, character of the pulse, and respiratory effort are also important parameters to assess and record.
- Sleeping respiratory rate (SRR) is commonly used in patients with cardiac disease as a sign of developing congestive failure. In healthy dogs SRR is 14 ± 3 pm; and in cats a mean of 19 pm [range 14–31].

TEMPERATURE

- Generally, 38–39°C (100.5–102.5°F) is the normal range, but this can increase to 39.5°C in stressed patients and rise to 40.5°C after exercise.

Table 1.6a Expected heart and respiratory rates in young and adult cats and dogs

(Consulting room)	Adult dog	Puppy	Adult cat	Kitten
Heart rate (range/min)	70–160	70–220	120–200	140–240
Respiratory rate (range/min)	18–28		20–30	
	Tends to be higher in kittens and puppies or if the animal is stressed. Most cats at home at rest have rates less than 24/minute.			

Table 1.6b Reference ranges for key parameters in neonates

Age (days)	Rectal temperature (°C [°F])	Heart rate (bpm)	Respiratory rate (/min)	Recommended environmental temperature (°C [°F])
0–7	35.5±0.8 [96±1.4]	200–500	15–35	29.5–32 [85–90]
8–14	37.8 [100]	70–220	15–35	26.5 [80]
15–28	No data	70–220	15–35	26.5 [80]
29–35	Adult	70–220	15–35	21–24 [70–75]
>35	Adult	70–220	Adult	21 [70]

- If there is concern that the patient's hyperthermia is physiologic, the owner should be encouraged to take the rectal temperature at home.
- Unfortunately, ear-based temperature measurement has not proven reliable; microchip-based temperature sensors tend to underestimate core temperature. Axilla temperature most closely reflects rectal temperature.

1.3 DIAGNOSTIC TECHNIQUES

1.3.1 *Haematology* 1.3.2 *Biochemistry* 1.3.3 *Other laboratory tests* 1.3.4 *Hormonal tests* 1.3.5 *Genetic tests* 1.3.6 *Urinalysis* 1.3.7 *Sample collection and preparation* 1.3.8 *ECG recording* 1.3.9 *ECG interpretation* 1.3.10 *Radiographic tips* 1.3.11 *Thoracic radiography* 1.3.12 *Abdominal radiography* 1.3.13 *Abdominal ultrasound* 1.3.14 *Echocardiography* 1.3.15 *Computed tomography*

INTRODUCTION

- This section covers the most frequently employed diagnostic techniques used in veterinary practice. Further information on the interpretation of specific tests is given in Section 3. For additional reference material see page 197 and the *BSAVA Guide to Procedures in Small Animal Practice* 2nd Ed. (2014).
- A good relationship with any laboratory used is important; both parties should be prepared to discuss the interpretation of results.

✪ Good case information aids the laboratory in interpreting the results.

- Deciding whether to use in-house or an external laboratory to measure blood parameters depends on a number of factors including range of tests, reliability of results, and time constraints, e.g. pre-operative assessments.

LABORATORY TESTS

1.3.1 HAEMATOLOGY (TABLES 1.7 AND 1.8)

- Precise reference ranges will vary between laboratories and in-house machines: breed- and age-specific variations also occur.
- Moderate to marked changes are more likely to be clinically significant and relate to a primary disease process. Mild changes (especially in older animals) are common, may be difficult to explain and may reflect disease elsewhere.
- Haematocrit (HCT) from most automated machines (derived parameter = RBCC × MCV) approximates to PCV. It can be significantly higher than PCV if the red-cell count is elevated, or in postal samples where red cell swelling raises MCV.
- Reticulocyte counts: used to evaluate an appropriate bone marrow response to the level of anaemia. Regenerative response in mild–moderate anaemia >100,000 (dog), 65,000 (cat)/µl; severe anaemia reticulocyte >250,000 (dog), 175,000 (cat)/µl.
- Low reticulocyte count can occur due to extra marrow factors, e.g. iron deficiency or insufficient response time (pre-regenerative).
- In leukaemia or leukaemic lymphoma, white cell count and the cell line affected can increase to >100,000/µl.

1.3.2 BIOCHEMISTRY

- Spun, separated serum is preferable to heparinised plasma for external biochemical analysis (see also 'blood sample types/tests' Table 1.10).
- Patients on potassium bromide have very high chloride measurements due to cross reactivity; check patient medication before interpretation.
- Low calcium may be due to chelation by ethylenediaminetetraacetic acid from haematology tube.

1.3.3 OTHER LABORATORY TESTS

- In addition to biochemistry and haematology, other tests may be appropriate when presented with patient signs suggesting:
    - ***GIT disease***: full faecal analysis, vitamin B12 and folate, trypsin-like immunoreactivity (TLI), pancreatic-specific lipase, basal cortisol or adrenocorticotrophic hormone (ACTH) stimulation test (to rule out atypical hypoadrenocorticism as a cause of GI signs), iron status if anaemic.
    - ***Renal disease***: urinalysis including urine protein:creatinine ratio and culture estimate GFR – symmetric dimethyl arginine (SDMA), iohexol clearance, erythropoietin level, acid/base status.
    - ***Liver disease***: dynamic bile acid testing, blood ammonia, bile cytology and culture, clotting profile.
    - ***Cardiac disease***: cardiac troponin I, NT-proBNP, taurine.
    - ***Anaemia***: reticulocyte count, erythropoietin, iron, total iron binding capacity, and transferrin.
    - ***Unexplained lymphocytosis***: flow cytometry.

1.3.4 HORMONAL TESTS

- Many hormones, e.g. parathyroid hormone (PTH) or ACTH, require careful handling, as they are easily destroyed during sampling or postage; check requirements.
    - ACTH stimulation test for hyper- and hypoadrenocorticism: measure basal cortisol, inject tetracosactide (Synacthen) IV or IM and measure cortisol after 45–60 minutes.
        - For a dog weighing >10 kg use 0.25 mg (1 vial); weighing <10 kg, use a half-vial.
        - Cats should be sampled at 1 and 3 hours, use a half-vial tetracosactide.

Table 1.7 Reference intervals for key haematologic parameters in adult and neonatal cats and dogs with interpretation of magnitude of change outside the reference interval

Parameter	Low			Reference	High			
	Marked	Moderate	Mild		Mild	Moderate	Marked	
Packed cell volume (PCV) (%)	15 12	23 18	30 22	37–55 27–50	60 55	65 60	75 70	dog cat
Red blood cell count (RBCC) ($\times 10^{12}$/l)	2.0 1.7	3.5 3.4	4.8 5.2	5.0–8.5 5.5–10.0	9.0 11.0	10.0 13.0	11.0 15.0	dog cat
Haemoglobin (Hb) (g/dl)	3.3 3.0	7.5 5.5	10.0 7.0	12.0–18.0 9.0–17.0	20.0 18.5	22.0 20.0	25.0 23.5	dog cat
Mean cell volume (MCV) (fl)	50 32	55 37	58 39	60–80 40–55	81 56	83 59	87 62	dog cat
Mean cell haemoglobin (pg)	22	27	30	31.5–37.0	NA	NA	NA	dog/cat
Platelets ($\times 10^{9}$/l)	10	50	120	160–600	650 700	800 1000	1000 1500	dog cat
WBCC ($\times 10^{9}$/l) (excludes leukaemia)	1.5 1.0	3.0 2.0	5.0 3.5	6.0–15.0 4.0–15.0	20.0 20.0	30.0 30.0	40.0 40.0	dog cat
Neutrophil	1.0 1.0	2.0 1.8	2.8 2.3	3.0–11.5 2.5–12.5	15.0 15.0	25.0 25.0	35.0 35.0	dog cat
Lymphocyte	0 0	0.5 0.7	0.9 1.3	1.0–4.8 1.5–7.0	5.5 7.5	7.0 9.0	10.0 12.0	dog cat
Monocyte	NA	NA	NA	0–1.0	1.5	3.0	5.0	dog/cat
Eosinophil	NA	NA	NA	0–1.3	1.8	3.0	5.0	dog/cat

Table 1.8 Reference intervals for key biochemical parameters in adult cats and dogs with interpretation of magnitude of change outside the reference interval

Parameter	**Kittens (mean or range)**				**Puppies (mean ± standard deviation, or range)**			
	0–3 days	2 weeks	4 weeks	6 weeks	0–3 days	0–2 weeks	2–4 weeks	6 weeks
PCV (%)	41.7	33.6–37.0	25.7–27.3	26.2–27.9	46.3±8.5	33–52.5	27–37	34
Hb (g/dl)	11.3	11.5–12.7	8.5–8.9	8.3–8.9	15.8±2.9	14–17.5	8.5–11.6	9.59
MCV (fl)	81.6	65.5–69.3	52.7–55.1	44.3–46.9	94.2±5.9	89–93	78–83	–
WBCC ($\times 10^9$/l)	7.55	9.1–10.2	14.1–16.5	16.1–18.8	16.8±5.7	16.8–23	23–25.5	15.00

- Low dose dexamethasone suppression test (LDDST) for hyperadrenocorticism (HAC): give 0.01 mg/kg (dog) 0.1 mg/kg (cat) IV dexamethasone sodium phosphate, and measure at 0, 3 or 4 and 8 hours.

✪ For accuracy, dilute to 0.2 mg/ml solution with water for injection and use an insulin syringe.

- High dose dexamethasone suppression test (HDDST) rarely used to distinguish pituitary from adrenal dependent HAC; measure endogenous ACTH instead (high in pituitary dependent hyperadrenocorticism (PDHAC) and low in adrenal dependent hyperadrenocorticism (ADHAC)).
- TSH stimulation test for hypothyroidism: recombinant human TSH 75 µg IV and measure thyroxin at 0 and 6 hours.

- See endocrine system (3.11) for further information.

1.3.5 GENETIC TESTS

- See section 1.1.5

1.3.6 URINALYSIS

- ***Definitions***: hyposthenuric = SG <1.008; isosthenuric = 1.007–1.015; hypersthenuric = >1.015 (Table 1.9).
- ***Collection***: catheter samples are rarely justified.
  - For cats, free catch using polystyrene packing, plastic beads (e.g. Katkor), non-absorbent 'sand', or washed fish-tank gravel.
  - For dogs, mid-stream urine flow is preferred.
- ***Cystocentesis*** allows accurate full urinalysis – send in a plain tube.
  - If there is gross haematuria on urination, compare a cystocentesis sample with a free catch, as this will help localise the area that is bleeding.
- ***Dipsticks*** are designed for humans: SG, leucocytes, protein, nitrite and urobilinogen can be unreliable or of no value.
- SG should be measured with a refractometer (Figure 1.3) – read from the right-hand scale as illustrated below for urine specific gravity (USG) (1.036) and from the middle scale for total protein (TP = 5 g/dl or 50 g/l).
- The refractometer should be calibrated periodically with water so the blue line reads at 1.000 (arrow Figure 1.3).
- ***Sediment***: should be inactive – few cells and no casts.
  - More than one or two casts per low power field usually indicates damage to renal tubules.
  - Presence of bacteria reported in sediment does not confirm urinary infection (low specificity).
    - Infection should be confirmed by culture as misidentification rate is high.

Table 1.9 Urinalysis parameters in dogs and cats

	Specific gravity (SG)		pH	Bilirubin
	Normally Hydrated	**Possible Range**		
Cat	>1.035	1.001–1.080	5.5–7.5	Negative
Dog	>1.025	1.001–1.065	5.5–7.5	+ → ++ in male

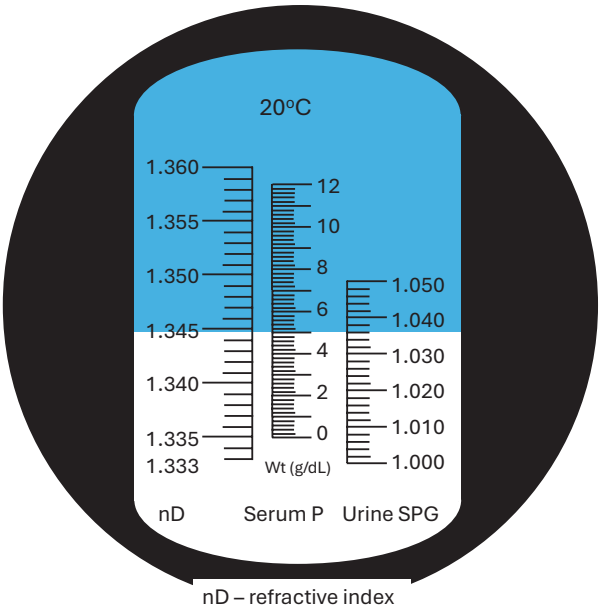


Figure 1.3 Scales visible when using a refractometer.

- ***Culture***: positive on cystocentesis, or $>10^5$ cfu/ml on free catch/catheter, would be significant results.
- NB interpret with clinical presentation as occult bacteriuria does occur (1–2% of samples in healthy cats and dogs).
- ***Urine protein:creatinine ratio*** gives a better estimate of proteinuria than dipstick result:
  - ratio of <0.2 is considered normal and >0.4 (cats) and 0.5 (dogs) abnormal.
- ***Urine cortisol:creatinine ratio*** can be a useful negative rule out for hyperadrenocorticism – sample collected at home.
  - For dogs the ratio should be $<30 \times 10^{-6}$.
  - For cats the ratio should be $<36 \times 10^{-6}$.

1.3.7 SAMPLE COLLECTION AND PREPARATION

- Correct sampling and handling are vital to obtain valid results.
- ***Blood samples***: a 0.5 ml sample (after red cells removed for serum and plasma) is usually sufficient; see Table 1.10 for those tests which need more.
- ***Urinalysis***: ideally a sample >2 ml. Only use boric acid tube if free-catch for culture.
- ***Fine needle aspiration biopsy (FNAB)***: 25–32 mm (1–1.25 inch) 23 g needle suitable for most samples (22 g spinal needle if more length required); take multiple samples.
  - Use a needle-insert technique without suction for soft masses/lesions, spleen, liver, or lymph nodes. Use suction with a 5 ml syringe for harder, less vascular structures.
  - Carefully smear using a spreader slide at 90° using only surface tension and no downward force.
  - Quickly dry in air or with hairdryer.
  - Remember to release the suction pressure before removing the needle to prevent material being lost into the syringe.
  - DO NOT store in the fridge or in the same package as formalin.

Table 1.10 Recommended blood tubes for various haematologic and biochemical tests	
EDTA	Haematology, fibrinogen, Coombs' testing, genetic and PCR testing (e.g. FeLV, FIV, *Mycoplasma haemofelis*)
EDTA plasma	Plasma osmolality
Frozen EDTA plasma	Endogenous ACTH*, PTH, PTH-RP*, renin*, NT-proBNP
Heparin plasma	Carnitine (frozen), ionized calcium, in-house laboratory analysis, can be used for external samples, plasma amino acids (frozen)*
Serum§	Most biochemistry, hormonal tests, antibody titres, drug levels, erythropoietin*
Citrate	Clotting times, anti-thrombin 3, D-dimer, von Willebrand factor, lead
Oxalate/fluoride	Glucose

* sample greater than 0.5 ml required.
§ separated serum (decanted or gel tube) much more reliable than whole blood.

- ***Biopsy***: try to gain material from advancing edge of a lesion/mass.
  - Ensure adequate formalin is used (10 × volume of biopsy).
  - If the biopsy is large, section into 10 mm strips.

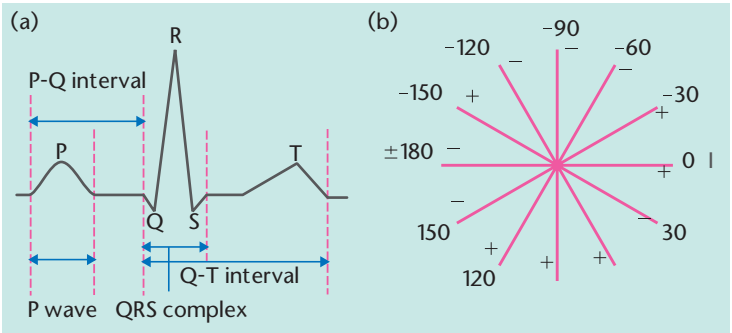
ECG BASCIS

1.3.8 ECG RECORDING

- A calm, quiet room is required.
- Patient should preferably be in right lateral recumbency, but if purpose is primarily rhythm it is better to allow the patient to adopt a comfortable position, e.g. in their basket for a cat.
- Chest leads are rarely required or of value. Electrode placement:
  - Red – right fore.
  - Yellow – left fore.
  - Green – left hind.
  - Black – right hind.
- Maximise complex deviation by turning up gain.
- If baseline artefacts are present, turn off electrical equipment in the room and use the filter options.
- Record leads I, II, and III at 25 and 50 mm/sec; aVR, aVL, and aVF at 25 mm/sec.

1.3.9 ECG INTERPRETATION

- Diagnostic quality: can complexes be clearly identified?
- Rate: consider overall rate as well as beat–beat variation.
- Rhythm: is it sinus (P for every QRS and QRS for every P)? (see Figure 1.4a)
- Every QRS must be followed by a T wave.
- Mean electrical axis: angle of lead at 90° to the isoelectric lead (sum of positive and negative deviation ≈ 0°) (Figure 1.4b).
- If abnormal beats or artefact(s) are present: how many? and are they all the same?
  - Supraventricular beats tend to be narrow and similar to normal QRS complexes; ventricular beats tend to be wide and bizarre.
- Measure complex deviation and time intervals (Table 1.11 and Figure 1.4).
- What might other deviations from baseline be?



Parts of the P-QRS-T complex. **Bailey's hexaxial lead system.**

Figure 1.4 (a) Parts of the P-QRS-T complex. (b) Bailey's hexaxial lead system.

Table 1.11 Reference intervals for P-QRS-T complexes in cats and dogs

	P wave	P-Q interval	QRS	Q-T interval
Dog	<0.04 sec <0.4 mV	0.06–0.13 sec	<0.05 sec <0.06 giant <3.0 mV	0.15–0.25 sec Normal heart rate (HR)
Cat	<0.02 sec <0.4 mV	0.05–0.09 sec	<0.04 sec <0.9 mV	0.12–0.18 sec Normal HR

RADIOGRAPHY

1.3.10 RADIOGRAPHIC TIPS

- Use positioning aids and sedation/anaesthesia as appropriate.
- In lateral view, use a wedge to raise the sternum parallel to the spine.
- Inflated anaesthetised films give most detail of the lungs, but don't overinflate.
- For lung views, maximise kV and minimise mAS to reduce movement blur.
- Deep-chested dogs may need separate cranial and caudal abdominal views.
- Obtain at least two views (left and right lateral, or right lateral and DV/VD of the chest; right lateral and VD of the abdomen).
- VD views in dyspnoeic patients can be dangerous.
- Use the windowing feature on image software to highlight different structure – can usually change intensity and contrast.
- For orthopaedic views: remember that soft tissue is important.
- Flexed and extended views of joints may be needed.
- For spinal films: try to keep the spine parallel to the plate.
- Skull radiographs can be difficult to interpret because of overlying structure, position head to highlight the structures of interest may require oblique, open mouth or skyline view.

1.3.11 THORACIC RADIOGRAPHY

Figures 1.5 and 1.6 illustrate normal thoracic radiographic anatomy.

1.3.12 ABDOMINAL RADIOGRAPHY

- At least two views of the abdomen are essential in order to locate the position of any abnormality. Usually these are a right lateral and VD, as in the diagrams.
- Note species difference: pylorus is more midline in cats than dogs.

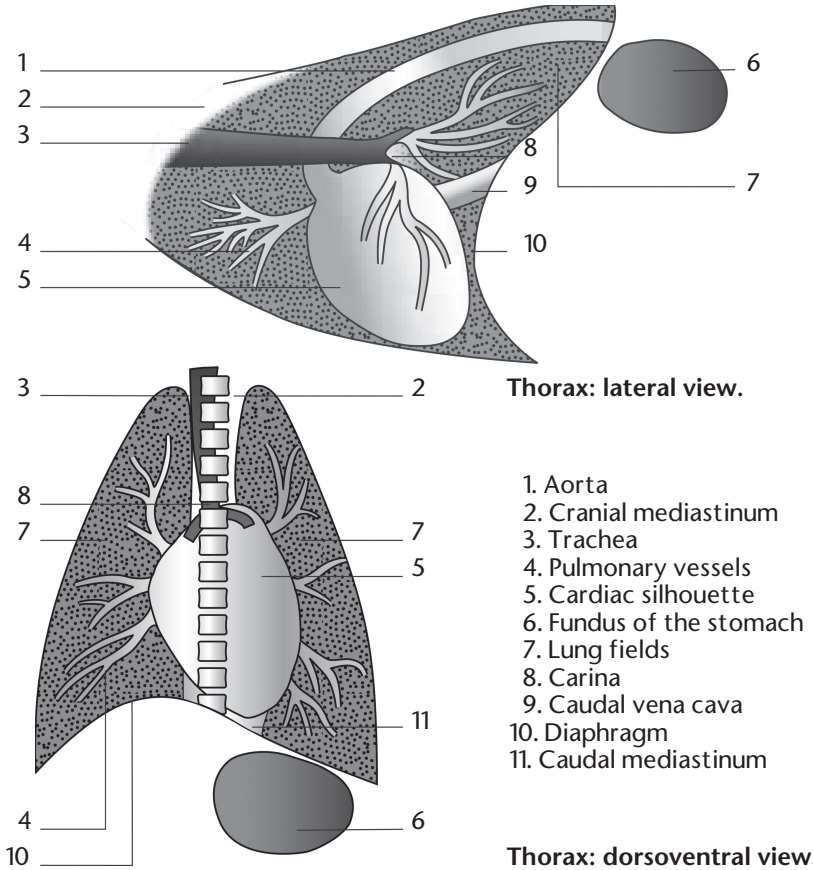


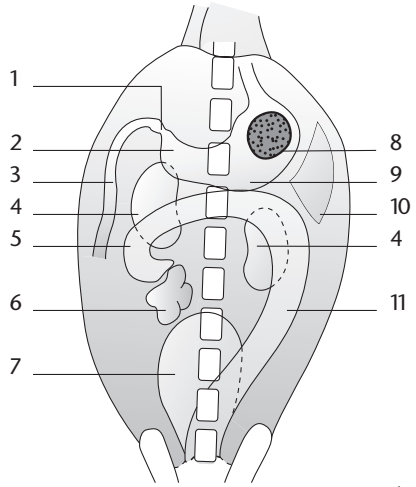
Figure 1.5 Radiographic anatomy of the thorax.

For complete evaluation of the stomach, four views are required; in normal cats and dogs, gastric gas is located as follows:

- VD view – fundus.
- DV view – body.
- Left lateral view – antrum.
- Right lateral view – fundus and body.

ULTRASOUND BASICS

- Full ultrasound of the abdomen, thorax, and other structures is a specialist technique that requires a lot of knowledge, understanding, and experience of normal variation and the interpretation of abnormalities, as well as appropriate equipment.
- However, the necessary skills can be acquired relatively easily to answer key questions that help with problem solving a case, e.g. the presence of free abdominal fluid or evidence of left atrial enlargement.
- When performing an ultrasound examination, it is important that the areas examined, limitations, and findings of the examination are recorded.



Abdomen: ventrodorsal view.

1. Pylorus
2. Antrum
3. Duodenum
4. Kidneys
5. Ascending colon
6. Caecum
7. Bladder
8. Fundic gas
9. Body of stomach
10. Spleen
11. Descending colon
12. Liver

Abdomen: right lateral view.

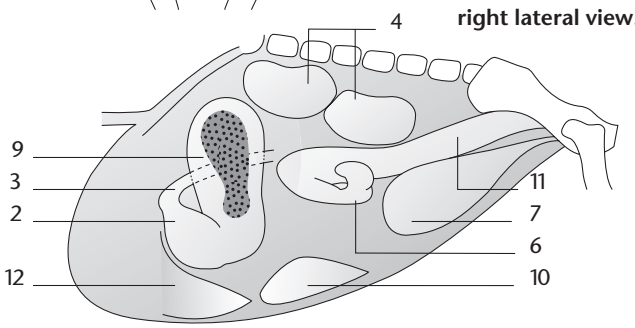


Figure 1.6 Radiographic anatomy of the abdomen.

1.3.13 ABDOMINAL ULTRASOUND

- Good patient preparation is important. Sedation may be necessary in some cases but can be minimised by a suitably comfortable table and quiet surroundings.
- A systematic approach should be adopted and abnormal findings described and then interpreted.

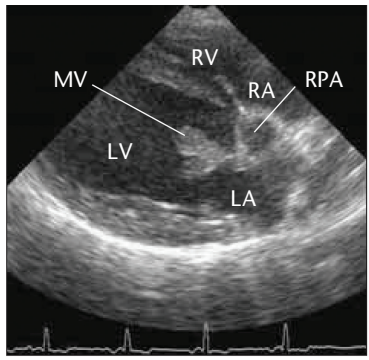
A suitable reference text will be helpful in illustrating probe positioning and normal variation. Knobology – correct machine settings can also make a significant difference. Settings are likely to need changing during examination to optimise the image of the target area.

1.3.14 ECHOCARDIOGRAPHY

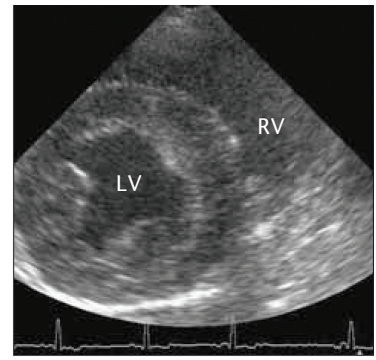
- Imaging the patient in lateral recumbency from underneath, via a cut-out in the tabletop, produces the best images.
- Phased array probes are preferred as it is easier to get a good image window between the ribs.
- A microconvex probe can be used for 2D, M mode, and some Doppler functions.

  Full cardiac ultrasound is complex, time consuming, and requires significant experience. However, a right parasternal approach to produce 2D short- (SA) and long-axis (LoA) views can generate useful decision-making information (Figure 1.7).

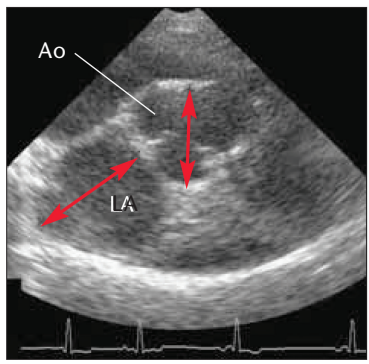
  - Long axis: ultrasound beam perpendicular to the long axis of the body and parallel to the left atrium of the heart showing the four chambers. A left



Long-axis view



Short-axis view



Short-axis view: aorta

Echocardiography. Standard right parasternal long and short axis views of the heart of a Cavalier King Charles Spaniel with mitral valve disease.

RV = right ventricle; LV = left ventricle; RA = right atrium LA = left atrium; RPA = right pulmonary artery; Ao = aorta; MV = mitral valve

Figure 1.7 Standard right parasternal long and short axis views of the heart of a Cavalier King Charles spaniel with stage B1 myxomatous mitral valve disease: (a) Long-axis view, (b) Short-axis view: left ventricle, (c) Short axis view: aorta.

ventricular outflow view showing the aortic valve is created by cranial angulation or clockwise rotation of the probe.

- Short axis: rotate transducer by 90° clockwise trying to produce a left ventricle with circular symmetry and an internal mushroom shape (created by the papillary muscles).
- Progressive dorsal angulation and slight clockwise rotation will bring the aorta into view.

✪ If it's round and central, it's probably the aorta.

KEY QUESTIONS

- Is there a pericardial effusion – a separation of the myocardium from the pericardium by hypo/anechoic fluid?
- Is there volume overload of the left heart – an enlargement of the left atrium (most easily assessed in short-axis view)
- What does the myocardium look like and how well is it moving?

MEASURING THE LEFT ATRIAL SIZE

- The ratio between the left atrium and aorta in diastole just before the aortic valves open is relatively unaffected by patient size.
- Ratio of less than 1.4 is normal and over 1.6 abnormal.
- The aortic measurement is taken when the valve is closed along the closure line between the non-coronary and right cusps. The left atrium is measured parallel from the line between the non-coronary and left cusp.

ASSESSING THE LEFT VENTRICLE

- In normal average-sized cats, interventricular septum or LV free wall thickness is ≤5.5 mm and LV diameter in diastole ≤19 mm.
- In dogs ≤15 kg a normalised ratio for LV diameter in diastole (LVIDdN) has been calculated at <1.7 LVIDdN = LVIDd (cm)/BWt (kg)$^{0.294}$.

ADVANCED IMAGING

MRI remains primarily a modality used at referral centres, but CT is becoming increasingly available in primary care practice.

MRI

Advantages	***Disadvantages***
No ionising radiation	Expensive
Excellent soft tissue contrast	Slower than CT thus more motion artefact
Differentiates fluid	Limited utility for cardiorespiratory disease
Can acquire images in any plane	Artefacts with metallic implants
Extensive disease applications	Pacemaker or metallic ocular FBs prohibit use

CT

Advantages	***Disadvantages***
Fast	Ionising radiation
Less expensive than MRI	Poorer contrast resolution vs MRI
Multi-planar reconstruction possible	Poorer fluid contrast
Reduced risk of motion artefact	Older models poor reconstruction
Extensive disease applications	Artefacts with metallic implants

1.3.15 COMPUTED TOMOGRAPHY

Computed tomography (CT) is particularly valuable for imaging the head (Figure 1.8) (limited brain detail), skeleton, and lungs. For the abdomen it depends on the question being asked but is generally superior to ultrasound in dogs over 25–30 kg. However, intestinal detail is poorer and, if changes are noted, CT guided aspiration is more complex.

Costs and risks are significantly greater than ultrasound.

Echocardiography is superior to CT for cardiac assessment.

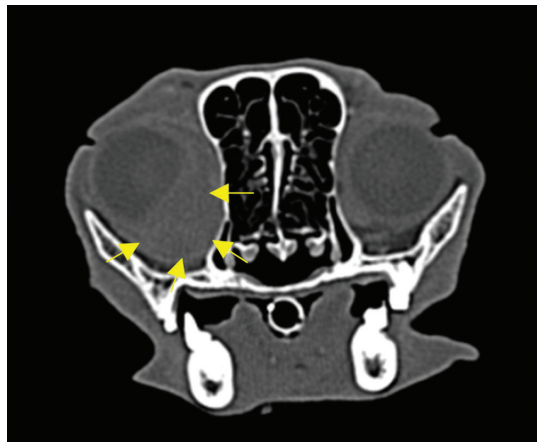


Figure 1.8 Retrobulbar lymphoma in an eight-year-old Mn DSH cat (arrows).

1.4 GUIDE TO COMMON PROCEDURES

1.4.1 *Abdominocentesis and thoracocentesis* 1.4.2 *Barium studies* 1.4.3 *Non-invasive blood pressure measurement* also Section [3.3.8] 1.4.4 *Blood transfusion* also Section [5.1] 1.4.5 *Cystocentesis* 1.4.6 *Gastric decompression* 1.4.7 *Gastric decontamination* reference to Section [5.4] 1.4.8 *In-saline agglutination test* 1.4.9 *Intravenous catheter* 1.4.10 *Myringotomy* 1.4.11 *Pericardiocentesis* 1.4.12 *Platelet count* 1.4.13 *Prostatic wash* 1.4.14 *Skin punch biopsy* 1.4.15a *Urethral catheterisation (male)* 1.4.15b *Urethral catheterisation (female) Techniques covered elsewhere* Arthrocentesis – Section [4.4.6]; Cardiopulmonary resuscitation – Section [5.9]; Fine needle aspirate biopsy – Section [1.3.7] Naso-oesophageal tube placement [1.1.10]

1.4.1 ABDOMINOCENTESIS AND THORACOCENTESIS

Diagnostic (sample analysis) or therapeutic (drainage).

Unless viscous fluid, e.g. pyothorax, 23 g needle for sampling, larger needles, preferably butterfly catheters, more appropriate for drainage.

RISKS AND CONTRAINDICATIONS

- Bleeding – increased if coagulopathy.
- Damage to organs – risks increase with blind sampling or if fluid volume is small. Puncture of GIT.
- Creating a pneumothorax.
- Appropriate sedation or anaesthesia reduces the risk of bleeding/organ damage but has its own risk profile and costs.

PATIENT PREPARATION AND POSITIONING

- Sternal or lateral recumbency – whichever gives best access and minimises risk, e.g. less respiratory compromise if large volume pleural effusion if patient sternal.
- Ultrasound guided wherever possible as reduces risk of organ damage, ensure fluid is sampled vs organ sampling or inadvertent cystocentesis. For a large effusion allows a suitable window for blind aspiration best approach to be identified.
- Clip area.

- For routine sampling then as for placement of IV catheter.
- For drainage sterile preparation ± fenestrated drape ± sterile gloves.

TECHNIQUE

Blind abdominocentesis – 10 mm behind umbilicus.

Blind thoracocentesis – 7–8th intercostal space at costochondral junction for fluid (Figure 1.9) or dorsal third for air; cranial aspect of rib.

- Insert needle at 90° to skin (local anaesthetic/cream as appropriate) with smooth motion and advance with syringe attached (preferably luer lock type) for thorax, ± syringe for abdomen. For larger volumes with dependent approach fluid will flow more freely without suction as less likely that omentum/viscera obstruct needle tip.

DRAINAGE

- Three way tap between needle and syringe or one way drainage extension.
- Spare arm connected to an extension set and taped into suitable sized bowl.

SAMPLE HANDLING

Always save a sample into EDTA and plain tube for analysis even if later discarded.

Analysis – total nucleated count, cytology, total protein, albumin and globulin, culture ± other, biochemical, serologic, or PCR tests.

1.4.2 BARIUM STUDIES

Barium sulphate is a radiopaque, non-osmotic, inert contrast medium useful for highlighting the intestinal tract. Barium impregnated polyspheres (BIPS) are available to evaluate gastric emptying, intestinal transit times, and detect obstruction, especially partial obstruction.

Barium can be given as a liquid, paste, or mixed with food.

Always take plain radiographs before giving barium as barium can obscure a radiodense foreign body (FB).

If chemical restraint is required acepromazine (0.01–0.02 mg/kg) is often effective.

Figure 1.9 Pleural drainage blood from rupture atrial mass.

RISKS AND CONTRAINDICATIONS

- Can be a skin irritant.
- Leakage into body cavity causes granulomatous reaction.

✪ *DO NOT USE IF GIT PERFORATION POSSIBLE*

- Care when administering orally that aspiration does not occur.
- Can cause constipation, transient diarrhoea, and abdominal pain.

INDICATIONS AND USE

- ***Oesophagus***: paste or barium mixed with food avoid liquid to reduce aspiration risk.

  Investigation of regurgitation, retching, dysphagia, gastro-oesophageal reflux.

  Voluntary eating/hand feed wet food formed into meat balls mixed with barium.

  Syringe (catheter tip) thick barium paste (60% w/v) @ 1 ml/5 kg to highlight mucosal abnormalities.

  Immediately obtain lateral thorax and cranial abdomen second radiograph 5–10 minutes later.
- ***Stomach and small intestine***: 30% w/v suspension @ 10 ml/kg.

  Investigation of persistent vomiting, haematemesis, suspected diaphragmatic rupture or other displacement of GIT, unexplained dilation or gravel signs (sediment radiodense material) of SI.

  If assessing stomach position/abnormality take four views immediately post administration.

  For gastric emptying and SI passage then films initially every 10–15 minutes.

  Once gastric emptying then every few hours, final image at 24 hours to show all barium in colon and exclude material FB.
- ***Large intestine***: 20% w/v suspension sufficient to fill LI (≈ 8 ml/kg)

  Investigation of tenesmus, haematochezia, chronic diarrhoea, suspected mass, or stricture.

  Ideally starve for 24 hours and warm water enema 2–3 hours beforehand.

  Patients can be sedated or anaesthetised.

  Place a foley catheter and instil barium per rectum– take VD and lateral view

  Drain barium and replace air for double contrast study.

1.4.3 NON-INVASIVE BLOOD PRESSURE MEASUREMENT

Most commonly used to assess for hypertension, e.g. in chronic kidney disease (CKD) or target organ damage, e.g. hyphaema that could be caused by hypertension

Monitoring cardiovascular function conscious or anaesthetised more commonly assessing for hypotension.

EQUIPMENT

Doppler or oscillometric system, suitable cuff (width 40% of circumference of leg/tail), quiet calm environment.

TECHNIQUE

Limb preferred to tail, non-weight bearing ideally at right atrial level.

- ***Doppler***: palmar arterial arch ventral aspect of metacarpal area, usually medially below carpal pad; plantar arterial arch ventral metatarsus; medial caudal artery ventral aspect of tail

  Primarily measures peak systolic pressure.

  Wear headphones, record limb used, cuff size, and patient attitude.

  Place cuff proximal to pulse, check inflates and stays on.

  Place Doppler probe over artery, fur may need to be clipped but for many patients liberal coupling gel is adequate and less stressful.

  Gently move probe until arterial signal detected and optimise signal.

  Inflate cuff until sound is lost; go 20 mmHg above this.

  Gradually deflate the cuff and listen for return of pulsation; the first sound gives systolic pressure and second sound diastolic (less reliable).

  Record six measurements, disregard first, and average the remaining five. Repeat if variability >20%.

- ***Oscillometry***: technically easier but failure to obtain measurements more common

  Place cuff as for Doppler measurement can also use brachial artery proximal to elbow.

  Initiate measurement – check heart rate displayed matches.

  Manual measurement.

  Record five cycles and calculate mean systolic, diastolic and mean pressure. Repeat if variability >20%.

- ***Reliability***: evaluate result in light of expectation. Inaccuracy can occur due to

  Inappropriate cuff size or placement; chondrodysplastic dogs may lack sufficient room for cuff to sit appropriately.

  Situational hypertension (white coat effect).

  Low blood pressure or arrhythmia.

  Excessive motion of limb or tail.

  Obesity, swelling, oedema at cuff site.

1.4.4 BLOOD TRANSFUSION

Key points on patient selection and preparation, calculation of need and basic administration and monitoring.

Blood bank products are variable available, in the UK canine packed red cells and various plasma products can be delivered usually same day. A number of labs offer cross-matching services. In-house blood group and cross-matching kits are available. Many practices keep a list of potential donors that are available to donate fresh, whole blood.

DONORS

Broadly donors should be fit and healthy, vaccinated, with regular parasiticide use and free from blood-borne infectious disease, e.g. FIV. Their blood group should be known. Ideally dogs should have a temperament that allows conscious donation: most cat donors will require sedation.

For a first-time canine recipient any donor can be used.

For cats it is essential that a blood group matched donation is given xenotransfusion – giving dog blood to cats should only be as a last resort.

Donation should be collected from the jugular vein 12–15 ml/kg for dogs and 11–13 ml/kg for cats is a safe volume.

Anticoagulant necessary 1 ml of citrate phosphate dextrose, citrate phosphate, dextrose adenine, or acid citrate dextrose per 7.3 ml of blood should be used.

For prefilled human collection bags, a minimum of 405 ml of blood is required (426 g), i.e. ≥28 kg dog.

For smaller dogs and cats collect into syringes or dry bags to which a suitable volume of anticoagulant has been added.

Accurate weighing scales are required to monitor flow and donation amount.

Sterile technique is important; critical if the blood is to be stored.

Donation should be gently agitated as it is collected to ensure mixing.

- ***Risks***: haematoma, hypovolaemic shock, aversion development.

RECIPIENTS

As blood is in relatively short supply, assessment of likely patient survival should be made.

- ***Indications***: haemorrhagic shock, non-regenerative anaemia, haemolytic anaemia. Usually wait until PCV in low teens or likely to reach that point in next 24–48 hours.

Whole blood may be necessary to manage coagulopathies, but plasma is better. In thrombocytopaenic patients, whole blood should only be given if there is active, uncontrolled bleeding as number of platelets in a donation is small with limited survival (maximum of 5 days usually less).

- ***Preparation***: for first transfusion in dogs blood grouping is not essential but should be performed if possible. Blood grouping in cats is essential. If there is sufficient time, then cross-matching to minimise the likelihood of a reaction is best practice. A cross-match should be performed in patients receiving a second or subsequent transfusion. Cross-match can be performed manually, with an in-house kit or externally.

  If the patient is very anxious or fractious, to protect the transfused blood from being wasted, sedation may be appropriate.

  Place a large-bore peripheral catheter and connect up the blood donation.

  If from a bag use a giving set with an in-line filter, if using blood collected in a syringe then use an 18–40 µm paediatric filter. Some syringe pumps can cause haemolysis of the blood so if in doubt use gravity flow.

  Calculate the amount of blood – roughly.

  Dog whole blood – 2 ml/kg raises PCV by 1%.

Dog packed red cells – 1 ml/kg raises PCV by 1%.

Cat whole blood – 2.5 ml/kg raises PCV by 1%.

- ***Administration***: initially at 1 ml/kg/hour for 30 minutes observing for transfusion reaction – fever, dyspnoea, tachycardia, vomiting, muscle tremor collapse, pruritus, oedema, haemoglobinaemia/uria

  After 30 minutes with no reaction increase rate to deliver remaining volume in 3–3.5 hours, monitor every 20–30 minutes.

  In an emergency situation, blood can be given at shock rates.

  Check PCV 3–4 hours after transfusion finished.

  Only 0.9% saline should be given through the catheter during transfusion.

1.4.5 CYSTOCENTESIS

Common procedure for obtaining a sterile urine sample, decompressing an overdistended bladder, comparing cystic to voided urine, e.g. for blood.

Risks and contraindications

Uroabdomen from puncture site, bladder rupture if severe disease, damage to adjacent structures, severe haemorrhage associated with aortic puncture, bladder too small.

TECHNIQUE

An appropriate length 23 g needle, sterile glass or plastic tube, 5–10 ml syringe. Clip and clean area for a non-surgical procedure.

Sedation may be necessary. The needle should be inserted with a smooth continuous movement.

- ***Blind***: (Figures 1.10a and b) cats and small dogs can be done standing from right side with bladder being pushed right and cranially. All patients in lateral or dorsal recumbency with the bladder located and pushed caudally towards pelvic brim.
- ***Ultrasound guided***: usually preferred as the bladder can be located and the risk of damage to surrounding organs minimised.

  Possible to obtain a sample from a smaller, non-palpable bladder.

  Easier for large or obese patients.

  In patients where the bladder is relatively small as the needle is pushed forward it can indent rather than penetrate through the bladder wall; in these cases, gently reduce the pressure and then make a short stab motion to penetrate through the bladder wall.

1.4.6 GASTRIC DECOMPRESSION

Stabilisation of dogs with gastric dilation and volvulus (GDV) that is severely impeding venous return and splinting the diaphragm.

RISKS

- Damage to the oesophagus and stomach including perforation by the tube.
- Needle damage to a displaced abdominal organ especially spleen.
- Haemorrhage.
- Intubation of the respiratory tract.

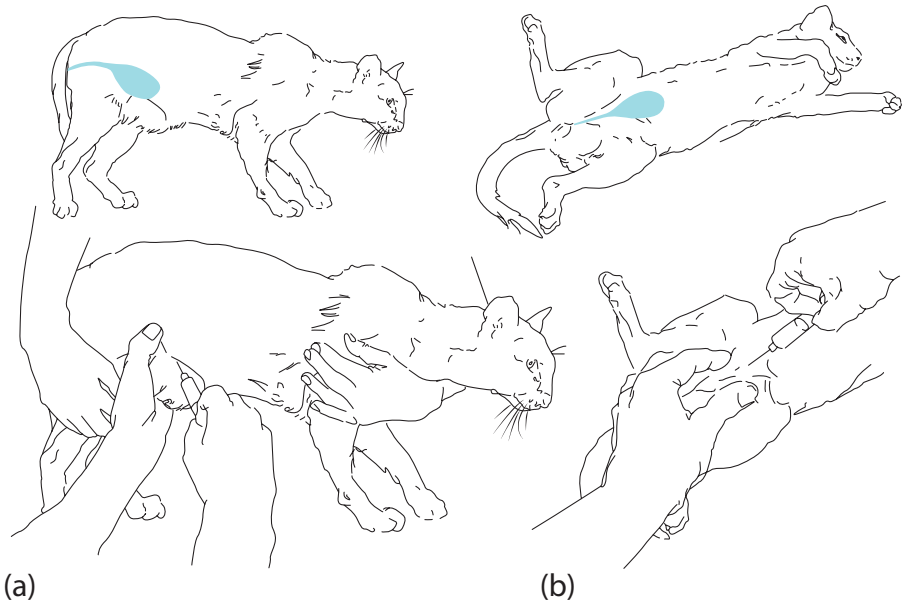


Figure 1.10 Blind cystocentesis in a cat: (a) standing lateral, (b) dorsal recumbent ventral approach.

PATIENT PREPARATION AND POSITIONING

- Drum-like abdominal distension associated with gastric dilation (radiograph if necessary, see Section 3.4.4).
- Patient sitting, sternal, or right lateral recumbency.
- Sedation may be necessary but care re hypotensive agents.
- IV catheter placement and provide fluid support, monitor ECG, and blood pressure.

TECHNIQUE

- ***Needle decompression***: clip and aseptically prepare an area midway up body wall caudal to last rib on left side (the bulging stomach can usually be palpated).
    - Insert a large bore needle or catheter (16–18 g) at 90° to the body wall directly into the stomach. Allow the gas to escape then remove the needle/catheter.
- ***Tube decompression***: measure a large bore tube to the 11th rib mark tube and do not insert beyond the mark.
    - Insert a bandage roll longitudinally in the mouth and pass a well lubricated tube through the centre.
    - At the gastro-oesophageal junction gentle tube rotation may facilitate passage into the stomach.
    - If entry into the stomach is not possible (signified by spontaneous decompression), do not force the tube decompress with a needle instead.
    - To fully empty the stomach, lavage may be necessary; tepid tap water can be used.
    - Pour in water from a height using a funnel and then drop the end below the table height, larger tubes are less likely to clog.

- ***Aftercare***: following decompression assess the cardiovascular stability of the patient and decide on next steps to manage the GDV.

1.4.7 GASTRIC DECONTAMINATION (SEE ALSO SECTION 5.4 POISONING)

Decontamination by emesis (more effective) or gastric lavage.

RISKS AND CONTRAINDICATIONS

Do not induce emesis if ingested substance(s) likely to contain:

- Caustic or corrosive substances as risk of further oesophageal damage.
- Volatile, petroleum, paraffin, or oily products as risk of inhalation.
    - Substances ingested more than 2–3 hours prior likely already in SI.
    - Perforation of damage gastric wall by stomach tube.
    - Post ingestion stricture.
    - Uncontrolled vomiting associated with emesis.

PATIENT PREPARATION AND POSITIONING

Emesis induction – patient standing/sitting and conscious.

Gastric lavage – lightly anaesthetised , intubated with a cuffed tube lateral or sternal recumbency, head below stomach.

TECHNIQUE

- ***Emesis***:
    - Sodium carbonate (washing soda) NOT CAUSTIC SODA – small crystal for cat/small dog and large crystal for medium-large dog.
    - α2-agonists – xylazine (0.6 mg/kg IM or 1 mg/kg SC) or (dex)medetomidine (10–20 μg/kg) IV/IM/SC usually in cats will cause sedation so care re aspiration
    - Apomorphine – 0.02–0.04 mg/kg IV (preferred) or SC; not recommended in cats.
- ***Gastric lavage***: technique as for gastric decompression.
    - (Section 1.4.6), lavage with warm saline or Hartmann's at 10 ml/kg.
    - Repeat (usually 10-–20×) until fluid appears clear.
- ***Activated charcoal***: reduces absorption of toxins; repeat doses every 4–6 hours required if enterohepatic recirculation, e.g. theobromines (chocolate), barbiturates, aspirin, and other salicylates.
    - Administer as liquid or slurry via stomach tube following gastric lavage.
    - Orally as slurry, if possible, otherwise tablets/capsules.
    - IF PATIENT VOMITS ACTIVATED CHARCOAL, IT WILL STAIN

1.4.8 IN-SALINE AGGLUTINATION TEST

Rapid test to determine if autoagglutination is occurring, suggesting immune mediated haemolytic anaemia.

TECHNIQUE

Add one drop of EDTA anticoagulated blood with one drop of 0.9% saline on a microscope slide and assess for agglutination both grossly (Figure 1.11a) and microscopically.

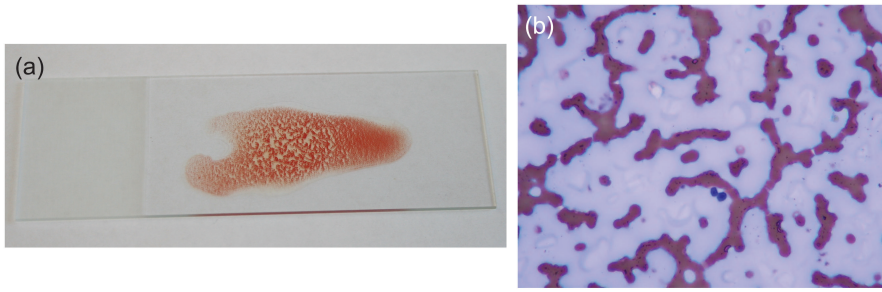


Figure 1.11 Assessing result of an in-saline agglutination test: (a) gross agglutination, (b) microscopic rouleaux formation.

Care must be taken to differentiate rouleaux (Figure 1.11b) (red cells stacking on top of each other) from agglutination.

If in doubt another drop of saline can be added.

1.4.9 PERIPHERAL INTRAVENOUS CATHETER PLACEMENT

Intravenous (IV) catheters are usually placed for the administration of fluids or drugs. Catheters may be short term (a few hours), e.g. during anaesthesia or longer term (up to 5 days), e.g. in sick hospitalised patients.

RISKS AND CONTRAINDICATIONS

- Catheters should not be placed at sites that are at high risk of or have existing infection.
- Care if patient is coagulopathic.
- Venous damage and thromboembolism.
- Phlebitis and thrombophlebitis.
- Air embolism.
- Exsanguination.
- Displacement and extravasation of fluid (rarely serious) or medications (potential for serious complications, e.g. perivascular sloughing).

PATIENT PREPARATION

- Site for placement should be clipped and aseptically prepared along with long hair on the other side of the limb.
- Sites – Cephalic and accessory cephalic vein.
    - Medial and lateral saphenous vein.
    - Auricular vein, e.g. Bassett.
- Pre-cut tapes, soft bandage, outer wrap and end cap or flushed T connector ready for use.
- Catheter selection dogs 18–22 g; cats 22 g; kittens/small puppies 24 g.
- Over the needle catheters most commonly used for peripheral veins.

TECHNIQUE

- Try and ensure the patient, placer and restrainer are comfortable.
- For some patients, sedation may be necessary – for planned procedures.
- Gabapentin or trazadone at home is often effective.
- Stabilise the limb and raise the vein.
- Palpate the vein and advance catheter through the skin at a 30–40° angle .
- Bevel upwards until blood is seen in the flash chamber.
- Reduce the angle between the catheter and skin and advance catheter whilst holding the stylet.
- Remove the stylet and bung the catheter.
- Tape the catheter in place and then connect an injection cap or T connector.
- Tape in and bandage leaving ports accessible.

CATHETER CARE

- Regular flushing (every 4–6 hours) with saline if catheter not being used.
- Examine catheter site for heat, erythema, swelling, pain, or leakage of fluid twice daily (unbandaging but not removing tape).
- If signs of phlebitis, then remove and replace at a different site: consider sending the catheter for culture.
- Regularly check the leg, foot and, toes for swelling that could indicate extravasation or that the bandage is on too tight (foot/toes).

1.4.10 MYRINGOTOMY

- To obtain a diagnostic sample to evaluate otitis media.
- Non-surgical management of otitis media via lavage of tympanic bulla(e).

RISKS AND CONTRAINDICATIONS

- Transfer of disease if otitis externa present.
- Neurological complications – head shaking, deafness, Horner's syndrome.
- Use of cerumenolytics where tympanum is not intact especially cats.
- Increases risks of neurological signs.

PATIENT PREPARATION

- Prior management of otitis externa (if present) to facilitate view of tympanum.
- General anaesthesia including intubation due to possible fluid drainage.
- from middle ear into pharynx.
- Muzzle down lateral (or sternal recumbency).
- Protect patient eyes if cerumenolytics used.
- Wear gloves, face mask, and eye protection.

TECHNIQUE

- Thoroughly clean and dry external canal – cerumenolytic agents may be used in dogs but all residual agent removed before myringotomy.
- Video or handheld otoscope used to image the tympanic membrane.
- Progress a small-bore catheter (3.5 Fr) along instrument channel or through speculum, the tip should be cut at an angle to create a sharp end to penetrate through the tympanic membrane
- Pass the tip through the membrane ventrally.
- Gently infuse 1ml sterile saline and aspirate for cytology and culture.
- Gently flush the middle ear until fluid returning is clear.

1.4.11 PERICARDIOCENTESIS

Relief of pericardial tamponade or sampling of pericardial fluid for diagnostic evaluation.

CONTRAINDICATIONS AND RISKS

- Suspected coagulopathy.
- Active pericardial haemorrhage or atrial rupture.
- Severe arrhythmia.
- Laceration of the heart or a cardiac tumour.
- Inadvertent cardiac puncture and removal of blood rather than pericardial fluid.
- Development of arrhythmias – common occurrence and rarely of clinical significance but can be life threatening.
- Cardiac arrest.
- Lung trauma and creation of pneumothorax.

PATIENT PREPARATION AND POSITIONING

- Sedation is usually required; place an IV catheter.
- Monitor the ECG throughout procedure.
- Sternal (by preference) or left lateral recumbency.
- Select and mark optimum window for procedure with ultrasound.
- Clip and aseptically prepare puncture site – usually 3rd–7th rib right side ventral half of thorax.

TECHNIQUE

- Infiltrate local at puncture site.
- Options for drainage will depend on availability and size of patient.
- Hypodermic needle – appropriate if sampling only; use ultrasound guidance.
- Small bore over the needle catheter – usually 18–20 G. Sufficient length to puncture the pericardium can be a problem in large, especially overweight, dogs.
- Large bore over the needle catheter – 10 G through which a 5 Fr urinary catheter can be passed.

- Small bore wire guided drain or pericardial drainage set.
- Usually easier to advance catheter through a small stab incision blind vs ultrasound guidance.
- Penetrate the pericardium (may require a sharp stabbing motion) – pericardial fluid is typically seen flowing in a pulsatile fashion; usually haemorrhagic.
- Connect to a three-way tap or one way drainage extension.
- Collect samples and drain as for thoracocentesis (1.4.1).
- Check the fluid does not clot – if clotting occurs check the catheter is not intracardiac.
- Check periodically with ultrasound to see how much fluid remains.
- Where there is only small amount left and the catheter tip irritates the epicardium arrhythmias can be induced; stop drainage at this point and back off or remove the catheter.
- Not all of the fluid needs removing to be effective – as the heart beats the catheter will have torn the pericardium so further drainage into the pleural space will continue to occur after catheter removal.

1.4.12 PLATELET COUNT

- A low platelet count is not uncommon on automated haematology. It is critical to patient management to decide if this is a real finding or artefact.
- Artefactual low platelet count can be caused by:
- Gross clot in sample.
- Platelet clumping.
- Presence of macrothrombocytes.

PREPARATION

- Fresh EDTA sample preferred but can use citrate or heparinised sample.
- Check that there are no gross clots in tube.

TECHNIQUE

- Make a blood smear.
- Check the feathered edge for platelet clumps (Figure 1.12a) – these can make the count inaccurate.
- Identify an area in the main body of the smear where there is a monolayer of red cells.
- Check for macrothrombocytes (Figure 1.12b).
    - Indicate a bone marrow response to recent blood loss.
    - Breed finding especially in Cavalier King Charles Spaniel but also Chihuahua, Labrador retriever, poodle, English toy spaniel, shih tzu, Maltese, Jack Russell, Havanese, Labradoodle, boxer, cocker spaniel, bichon frise, miniature Schnauzer, Belgian malinois/shepherd cross, komondor.

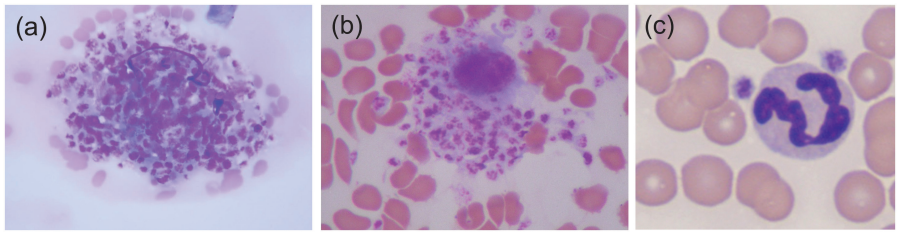


Figure 1.12 Assessing platelets on a peripheral blood smear (a) platelet clump, (b) macrothrombocytes, (c) normal platelets

- Count the number of platelets (Figure 1.12c) and average for 3–5 high power fields (40× objective). 1 platelet/hpf approximates to 6,000/µl (6×10^9/l).
- Reference interval ≥35/hpf.
- Thrombocytopenia consistent with haemorrhage ≥12/hpf.
- Immune mediated thrombocytopenia can lower platelet count to 0/hpf.
- Spontaneous gross haemorrhage unlikely if ≥5/hpf.

1.4.13 PROSTATIC WASH

Prostatic washing can help differentiate benign prostatic hyperplasia (BPH), prostatitis, and prostatic neoplasia. It can also be used when investigating pyuria or haematuria especially if changes are not present on a cystocentesis (1.4.5) sample.

CONTRAINDICATIONS AND RISKS

- Urethral trauma or rupture.
- Ascending urinary tract infection.
- Rupture of prostatic abscess.
- Rectal trauma/rupture.

PATIENT PREPARATION AND POSITIONING

- Light sedation is usually appropriate.
- Patient in lateral recumbency (left if operator right-handed).
- Clean prepuce with dilute hibiscrub or povidone, rinse off.

TECHNIQUE

- Gently advance a sterile catheter into the bladder (1.4.15a) and empty urine.
- Flush bladder out with saline, keep a sample to compare with prostatic wash fluid.
- Guide tip of catheter (remember holes are not at end) over prostate per rectum or with ultrasound.
- Massage the prostate per rectum or transabdominally for 1 minute.
- Inject 1–2 ml of sterile saline into urinary catheter.
- Repeat massage and then aspirate fluid back.

- Repeat injection/massage if insufficient sample.
- Make a direct smear, place some in EDTA (cytology) and a plain tube (culture).

✪ Differentiating dysplastic from neoplastic cells can be challenging.

1.4.14 SKIN PUNCH BIOPSY

- Investigation of unexplained dermatosis that has not responded to appropriate rationale therapy.
- Limited value in larger nodular lesions.
- Variable for ulcerated lesions depending on size.
- Consider other methods for junction between normal and abnormal skin such as an excision or incisional wedge biopsy.

CONTRAINDICATIONS AND RISKS

- Uncontrolled bleeding.
- Poor wound healing.

PATIENT PREPARATION AND POSITIONING

- Usually requires light sedation and local anaesthesia unless sensitive area such as face or feet.
- Cut hair at biopsy site with curved scissors.
- Minimal skin preparation – use 70% alcohol wipe if for culture.
- 6–8 mm biopsy punch unless restricted site (use 4 mm punch).

TECHNIQUE

- Infiltrate the skin and subcutis with local anaesthetic.
- Select the site of the lesion for biopsy – if there are multiple lesions then biopsy a number at different stages/sites.
- Press biopsy firmly on to skin and rotate in one direction into the subcutis.
- Lift the base with fine forceps and separate with curved scissors.
- Close the defect with a single suture or staple.
- For histology place biopsy subcutis down on card with an arrow in direction of skin growth. Use a separate sample pot for each labelled as to site in case histologies are different.
- For culture (bacterial or fungal) place in sterile container with a few drops of saline.

1.4.15A URETHRAL CATHETERISATION (MALE)

- Empty urinary bladder ± collect sample.
- Administer radiographic contrast.
- Indwelling:
  - Collect urine in recumbent patient.
  - Measure urine output.

- Keep bladder empty to allow tight junctions to reform.
- Maintain a patent urethra (short term).
- Allow urethral rupture to heal.

! If the patient is blocked, severe metabolic derangements may be present and should be stabilised prior to catheterisation. Cystocentese to empty the bladder.

CONTRAINDICATIONS

- Pre-existing urethral trauma.
- Urethral mass or stricture.

RISKS

- Urethral trauma or rupture.
- Introduction of infection.

PATIENT PREPARATION AND POSITIONING

- Most dogs can be catheterised with gentle restraint or light sedation.
- Most cats require sedation ± epidural or general anaesthesia.
- Area around prepuce may need clipping in long-haired breeds.
- Clean with dilute hibiscrub or povidone and rinse off.
- Use silicone foley for indwelling catheters in dogs and female cats.
- A guide wire is usually required to stiffen the Foley for placement.
- Polyamide catheters are inexpensive and relatively rigid they are useful for single catheterisation.
- A variety of tomcat catheters are available made from silicone, polytetrafluorethylene and polyurethane can be indwelling.

TECHNIQUE

- Select an appropriate sized catheter for the patient's size and sex (Table 1.12).
- With suitable restraint in lateral recumbency an assistant extrudes the penis.
- Male dogs – stabilise os penis with one hand and retract the prepuce caudally.
- Tomcats – extrude the penis by applying pressure each side of the prepuce.
- Lubricate the catheter tip well and feed from inside the sterile sleeve if possible, slowly advancing.

Once the catheter is at the caudal os penis (dogs); >25 mm (cats initially may require the prepuce and penis to be pulled caudally to straighten the membranous urethra) the penis can be allowed to retract into the prepuce.

Table 1.12 Guidelines for urinary catheter selection

	Dog	Bitch	Tomcat	Queen
Diameter (Fr)	5–10	5–16	3–4	4–5
Length (cm)	30–60	30–40	11–14	11–30

If the catheter meets resistance, then gentle flushing with sterile saline or rotation can help advancement.

Rarely a urethrolith will completely block the urethra and a catheter cannot be passed; urohydropulsion (dogs) or surgery may be necessary.

Once in the bladder and urine is flowing back then advance another 10 mm (cats); 10–20 mm (dogs) to ensure comfortably in the bladder before inflating the Foley cuff or suturing to the prepuce (tomcat).

ALWAYS place an Elizabethan collar or similar to prevent an indwelling catheter from being removed.

1.4.15B URETHRAL CATHETERISATION (FEMALE)

Indications, contraindications and risks are similar to males.

CONTRAINDICATIONS AND RISKS

Additionally, a risk of penetrating the cervix into the abdomen.

PATIENT PREPARATION AND POSITIONING

BITCH – moderate sedation is usually required. Recumbency depends on individual preference – dorsal with hindlimbs cranially or frog legged, lateral (digital palpation only) or sternal with legs over edge of table.

QUEEN – lateral or sternal (preferred as easy to keep catheter in the midline) recumbency.

TECHNIQUE

Blind catheterisation (all cats and some dogs) – keeping the catheter in the midline of the vestibule floor (ventral) progress gently cranially, the urethra is in a depression in cats and the catheter will naturally fall into it.

In dogs the urethral opening lies cranial to the clitoral fossa.

Digital palpation for large enough dogs, the urethral orifice (papilla) can be palpated as a slit on a slight 'bulge' of the mucosa.

Advance the finger just beyond the opening and applying pressure to the ventral floor slide the catheter under your finger to engage the urethral opening.

Direct view patient in dorsal or ventral recumbency, pass a lubricated sterile speculum into the vestibule avoiding the clitoral fossa (ventrally).

The urethral papilla lies dorsal and cranial to the clitoral fossa and ventral to the vagina.

The catheter can be passed directly into the urethra.

Dependent on breed and body condition, direct viewing of the urethral papilla even with a speculum can be challenging.

All

Once the catheter is in the urethra, advance into the bladder by 10–20 mm as for males and inflate the Foley. It is generally not recommended that a rigid catheter is left indwelling in female patients as security is poor and urine will often leak around the catheter.

ALWAYS place an Elizabethan collar or similar to prevent an indwelling catheter from being removed.

COLLECTION SYSTEM

A closed collection system is preferred as it reduces infection risks. If unavailable then bung the catheter with intermittent drainage.

Antimicrobial cover should not be used routinely.

1.5 THERAPEUTICS

1.5.1 *Making therapeutic decisions* 1.5.2 *Managing therapeutic disasters* 1.5.3 *Monoclonal antibody therapy* 1.5.4 *Key facts about the top 20 therapies used in primary care practice*

See also vaccinology [1.1.6], parasite control [1.1.7], poisoning [5.4], and antimicrobials [3.13.4–3.13.9]. See index for individual drugs. Organ specific therapies see Section 3. Anaesthesia and analgesia see Sections 4.1 and 4.2. Critical care drugs in Section 5.

1.5.1 MAKING THERAPEUTIC DECISIONS

Therapeutic decisions should be based on a number of factors:

- Will the owner be able to administer the medication to the patient?
- Efficacy.
- Potential risks to owner or owner's family.
- What type and level of monitoring is required.
- Licensed for the condition being treated vs licensed for that species vs licensed for veterinary use vs licensed for human use vs special treatment licence.
- Potential drug interactions with current therapies.

Always check with owners about other medication that they are currently giving, including over-the-counter products, herbal remedies, and supplements.

- Previous experience of using the drug in this patient.
- Known side effects and their likelihood.
- Cost.

When discussing medication options with the owner the above should be included in the final decision made with further written information provided as appropriate:

Essential information for the owner about any medication.

- Route of administration.
- Dose rate.
- Frequency – how evenly spread out should the doses be?
- If multiple medication which can be given together?
- Relationship with feeding – before, after, with food, anytime.
    - For many owners knowing whether oral medication that should not be given with food can be given with a small treat is important.
- Duration and timing of any dose change.
- Likely side effects and what to do if they occur.
- Side effects

It can be a difficult decision as to which and how many side effects to discuss. Generally, it is appropriate to inform owners of very common side effects (occur in >1 in 10 patients)

such as PU/PD associated with glucocorticoids effects that occur in between 1:10 and 1:100 patients. Side effects that occur in <1:100 patients if they are potentially severe.

1.5.2 MANAGING THERAPEUTIC DISASTERS

Severe medication side effects are rare but when they occur the most likely consequences are anaphylaxis, severe damage to the GIT including perforation, acute kidney injury, e.g. NSAIDs, hepatopathy, e.g. lomustine, erythema multiforme, e.g. cephalexin, neurologic side effects, e.g. seizure wih amitriptyline or ataxia with metoclopramide, collapse, e.g. hypotension with β-adrenergic blockers, and severe bone marrow suppression, e.g. azathioprine.

Potentially any drug can cause any side effects, but certain drugs are more commonly associated with specific issues.

The key is to:

- Recognise that the presentation may be drug related, e.g. post-surgical anorexia associated with AKI caused by NSAID therapy.
- Stop therapy with that drug as quickly as possible.
- Provide symptomatic and supportive treatment.
- Look for possible specific treatments, e.g. misoprostol with NSAID toxicity or granulocyte colony stimulating factor with bone marrow suppression. Some cases will require intensive care such that referral should be considered.

1.5.3 MONOCLONAL ANTIBODY THERAPY

Over the next decade or so, it is likely that a whole raft of monoclonal antibody (McAb) therapies will become available for use on cats and dogs.

McAb therapies are likely to be particularly useful in managing neoplasia, immune-mediated disease such as allergy, and chronic disease where switching the immune system on or off or specifically blocking the action of a molecule or enzyme are likely to be helpful.

A number of products have been licensed for veterinary use, e.g. for the treatment of allergic dermatitis in dogs and osteoarthritis (OA) in cats and dogs.

The key things to remember about these products are that:

- Usually species specific (unless inhibiting common pathway, e.g. Janus kinases) – use in another species risks both a lack of efficacy as the McAb may not recognise the target and could cause a significant immune response as the antibody is foreign.
- Target a specific receptor so they may have very limited action outside of the specific indication for which they were created.

By being more target specific, the effects should be more predictable and side effects less, which, to date has been the general experience of the veterinary products. This is also a potential weakness as if the pathway being targeted is not responsible for the clinical presentation the drug will not have a suitable action, e.g. only some dogs/cats with OA seem to respond.

Inherently this means that our diagnoses need to become more specific to effectively deploy these products.

1.5.4 KEY FACTS ABOUT THE MOST COMMON (NON-PARASITICIDE) ORAL THERAPIES USED IN PRIMARY CARE PRACTICE (TABLE 1.13)

Drugs in italics do not hold a license for use in cats or dogs.

Table 1.13 Key facts about the most common (non-parasiticide) oral therapies used in primary care practice

Medication	Frequency	Dose rate	Relation to food	Side effects and contraindications
Carprophen	q12–24 hr	4 mg/kg/day for 5 days then 2 mg/kg/day (D)	Any	Avoid in dehydrated, hypovolaemic or hypotensive dog, if there may be GI ulceration or severe cardiac, hepatic or renal disease
Meloxicam	q24 hr	0.1 mg/kg (D); 0.05 mg/kg (C)		
Gabapentin	q6–8 hr (D) q8–12 hr (C)	10–20 mg/kg (D); 5–10 mg/kg (C); start at lower dose	Any	Sedation and ataxia Rebound pain or seizures if rapid withdrawal
Cephalexin	q8–12 hr	15–30 mg/kg (D & C)	With food	Vomiting and diarrhoea – reduced by taking with food. Erythema multiforme (very rare)
Doxycycline	q24 hr	10 mg/kg (D & C)	With food	Nausea, vomiting, diarrhoea. Bound by antacids, calcium, magnesium and iron salts. Oesophagitis with hyclate salt – give 5–10 ml water after tablet
Metronidazole	q12 hr	10–15 mg/kg (D & C) 25 mg/kg max 5 days for Giardia treatment	Any	May be teratogenic, dose dependent neurotoxiciy. GIT side effects common. Glossitis and stomatitis. Hepatotoxicity, neutropenia and haematuria
Potentiated amoxicillin	q8–12 hr	12.5–25 mg/kg (D & C)	With food	Vomiting and diarrhoea – reduced by taking with food. Skin rash
Furosemide	q6–24 hr	Maximum dose 12 mg/kg/day	Any	Avoid if patient dehydrated, hypovolaemic or hypotensive. Can precipitate AKI or low output heart failure. PU/PD common. Nephro- and ototoxic
Pimobendan	q12 hr	0.2–0.3 mg/kg (D & C)	1 hr before or 2 after	Vomiting. Use with care in hypertrophic disease
Maropitant	q24 hr	2 mg/kg (D); 1 mg/kg (C) 8 mg/kg motion sickness	Before	Avoid if obstruction or perforation suspected Haemolysis in cats at high dose
Metoclopramide	q6–12 hr	2 mg/kg/day (D & C)	Before	Avoid if obstruction or perforation suspected. Do not use if GI haemorrhage or epilepsy. Reduce dose by 50% if significant hepatic/renal disease. Changes in mentation and behaviour especially cats

Omeprazole	q12 hr	0.8–1.3 mg/kg (D & C)	Before	Nausea, diarrhoea, constipation, skin rashes, and tooth fractures. Chronic use >8 weeks not recommended. Side effects can mimic reason for use
Chlorphenamine	q8 hr q8–12 hr	4–8 mg/dog 2–4 mg/cat	Any	Mild sedation and may reduce seizure threshold Avoid if GI obstruction, urinary retention, or glaucoma
Oclacitinib	q12–24 hr	0.4–0.6 mg/kg	Any	Diarrhoea, vomiting, anorexia, swellings, lethargy, polyphagia, polydipsia, proteinuria, aggression. May increase susceptibility to infection, demodicosis, and histiocytoma
Prednisolone	q12–24 hr	Depends on disease being treated. Maximum 2 mg/kg/day (D), 4mg/kg/day (C)	Any	Multisystemic actions, PU/PD and polyphagia very common. Do not use with NSAIDs or pregnant animals. Generally contraindicated in renal disease, congestive heart failure and diabetes mellitus. Chronic use iatrogenic hyperadrenocorticism
Phenobarbitone	q12 hr	Start @ 2.5–3 mg/kg (D) 1.5–3 mg/kg (C)	Any	Widespread interaction with other drugs. Sedation, ataxia, PU/PD and polyphagia common Avoid in patients with significant hepatic disease. Regular monitoring for level and side effects required
Phenyl-propanolamine	q8–12 hr	2.4 mg/kg/day (D & C)	Any	Sympathomimetic – some peripheral vasoconstriction Can cause restlessness, aggression, irritability, GIT signs and hypertension. Caution with CV, renal, hepatic disease, glaucoma, diabetes, hyperthyroidism

1.6 CLIENT COMMUNICATION AND PROFESSIONAL CONDUCT

1.6.1 *Client communication* 1.6.2 *Euthanasia* 1.6.3 *Dealing with difficult situations* 1.6.4 *Key points of professional conduct* 1.6.5 *Continuing professional development* 1.6.6 *Patient referral*

INTRODUCTION

Most complaints to practices and regulatory bodies centre around a failure of communication. Conversely good communication leads to higher levels of client satisfaction and better outcomes for patients. What you actually say accounts for 7% of the message, your tonality 38%, but, most importantly, body language (non-verbal communication) delivers the remaining 55% of the overall message.

1.6.1 CLIENT COMMUNICATION

Good communication requires greater time, effort, and structure the larger and more complex an organisation becomes. Good communication is not just about being nice or compliant with an owner's wishes, it is a professional skill that can be learnt and developed.

Keeping consultations on time – failing to keep to time is a major stressor for both clinician and owner and can be very challenging in a busy practice with fully booked appointments, especially where 'extras' have to be shoehorned into an already full schedule. Structuring the consultation and allowing time for each part is important; see Section 1.2.

Time in consultation can be saved by:

- As much preparation as possible prior to the consultation.
- Pre-consultation questionnaire, especially for new clients to capture basic details and information that may take time for a client to remember, e.g. diet.

If, during the initiation of the consultation it becomes clear that there are multiple issues to discuss, it may be appropriate to focus on one or two urgent problems and arrange for the remaining issues to be dealt with at another time.

Where minor procedures are indicated, e.g. blood sample, FNAB, BP measurement, consider whether they can be undertaken at the time or whether the patient should be admitted, or they need to be scheduled for a different day. Trying to rush a procedure during a busy consultation period, particularly with an anxious, nervous, or aggressive patient can often take far longer than expected causing further time pressure.

If consultations are running behind, then:

- Make sure clients that are waiting are kept up to date about the delay and given the option to reschedule.
- Ask if additional help is available to see clients.

Delivering the plan and discussing fees (see also 1.2.2)

Once the examination is completed the client should be presented with a plan – this could include a small number of differential diagnoses.

The plan should be discussed with the owner and their questions answered. This is usually an appropriate time to discuss fees, recognising that this can be an emotionally charged topic. Clients may feel guilty that they do not have unlimited finances and it is worth discussing the potential budget early on so that time is not spent discussing unaffordable options.

A clear understanding of the practice fee structure is critical to deliver an appropriately costed plan. Itemised estimates are helpful for some clients as they can see how the final cost is made up and also allows individual items to be discussed such as the value of pre-operative blood tests.

Regardless of how you feel, the estimate or account should be given without defensiveness or embarrassment or phrases like 'I know it is a lot of money but …'. Often a client will ask, 'if it was your pet what would you do?' You should be prepared to answer such a question as they are asking for your professional opinion, being evasive and using phrases like 'Well it's really your decision' are rarely helpful nor do they engender trust. Trying to answer in terms of the patient's health and welfare tailored to the pet–owner combination is usually appreciated.

Prognosticating and survival times

It is not unusual for clients with pets that have more serious or chronic disease to ask what their pet's prognosis is likely to be. Your answer can be part of their decision making in terms of what plan to follow and how much they are willing to spend. Prognosticating is fraught with difficulty, especially in acutely unwell patients where the diagnosis is uncertain.

Being honest about the level of uncertainty is important.

Where data are available, give rough percentage survival rates.

Where there are no data, or you do not know the answer, then give broad categories using language that the owner will understand.

Estimating likely survival times, even with common conditions such as mitral valve disease where reasonable amounts of data are available is difficult. Being over-optimistic or evasive is not advisable and can lead to future resentment. It is not unreasonable to say that it is difficult to be accurate especially if tests or procedures are pending. It should always be emphasised that expected survival times are an estimate and that some patients do better than expected and sadly some not as well as hoped.

Broad timeframes are usually the best option:

- Days to a few weeks.
- A few months.
- 3–6 months.
- 6–12 months.
- 1–2 years.
- >2 years/unlikely to affect life expectancy.

1.6.2 EUTHANASIA

Most veterinary professionals are 'fixers and problem solvers' by nature with a recommendation that euthanasia is the best option being seen as failure; this can result in suboptimal welfare. Many owners are also reluctant to introduce euthanasia as an option but are 'relieved' when it is included in possible approaches to their pet's problems.

Euthanasia can be very stressful for all parties. Ensuring that euthanasia goes smoothly is in the best interests of the patient and ensures the last memories that an owner has of their pet are the best they can be and is often a benchmark that owners use to assess the competence and professionalism of the personnel involved and practice in general. Many practices will have protocols and guidelines around euthanasia which should be followed as this delivers a consistent approach and means everyone is clear about their role.

- ***Preparation*** is key to success
    - Be clear about whether this is a discussion about euthanasia or whether the decision has been made and further discussion is not needed or would not be welcomed by the client.
    - Have there been discussions about payment and what the client would like to happen to their pet's body?

  - Know the patient in terms of temperament and reason for euthanasia and how these might impact on the procedure.
  - Know the client – how have previous euthanasias gone? Are there issues surrounding this particular case?
  - Make a plan, e.g. where is the euthanasia to take place, how will the clients leave the practice, will a catheter be placed, will sedation to be used? Discuss with all of the team involved.
  - Home euthanasia – check sufficient syringes, catheters, euthanasia solution, etc. Consider the logistics of performing the euthanasia, carrying the body, and transporting back to practice.
  - Have the consent form ready for signing.
- ***Procedure*** in most cases placing an IV catheter ensures the actual injection is incident free. Consider placing the catheter without the owner present.
  - Sedation can be helpful in anxious, nervous, or aggressive patients. Choose a combination that will produce good levels of sedation but has minimal impact on BP otherwise catheter placement can be difficult.
  - Prewarn the owner that their pet may lose bowel control and there may be post-euthanasia reflex diaphragmatic contraction causing gasping.
  - Make sure the owner is aware that grief support is available (some practices have in-house support), e.g. Cats Protection Paws to Listen, Blue Cross Pet Bereavement Support Service.
  - Once ready, give the owner time to say goodbye, flush the catheter to ensure patency (let the owner know it is flush), and then check the owner is ready before injecting the euthanasia solution.
  - Confirm with the owner the patient is dead, check whether they want to keep collar, take hair clippings or paw prints. Confirm disposal arrangements.
  - Allow the client time with their pet's body, ensure someone is available to walk them out of the practice; and if they are paying on leaving that they do not have to wait at reception.
  - Ensure any future consultations or reminders, e.g. for vaccination, are cancelled.
  - Many practices follow up with a sympathy card.

1.6.3 DEALING WITH DIFFICULT SITUATIONS

Dealing with difficult situations is part of client communication, requiring the same core skills and understanding. Managing these situations calls for emotional intelligence and a recognition of your own and a client's emotional state such as sadness, anger, or guilt and the intensity of those emotions.

Acknowledging that the situation is emotionally charged and reflecting back the emotion you perceive is the first step. 'I understand that you are angry and upset. What can we do to resolve this?'

It is particularly difficult when a client is angry and upset when you feel it is unjustified and you have already gone above and beyond to help them. Despite your emotions, you need to listen and allow the client to express their thoughts; try and remain empathetic and non-judgmental. Problem solving or defending actions at this point is rarely helpful. Try to summarise and reflect back the reason for the outburst and agree a way forward.

Summarise the conversation and actions on the clinical notes.

If you feel physically threatened, leave the room and seek help and support.

Handling complaints

Complaints may be verbal to you or another member of the practice of sufficient seriousness to require action, arrive by letter or email from a client or another practice, or come via the regulator.

Complaints are an inevitability of dealing with people and are not always from the situations from which you might expect a complaint to arise. All practices should have a complaints procedure which should be followed. Despite the natural instinct to view a complaint as unjustified and a tendency to put off dealing with it in the hope it will go away, this rarely happens and risks escalation.

When a complaint is received, it should lead to an:

- Acknowledgement that an event has occurred that needs addressing.
- Apology (not admission of guilt) that something has happened that has caused distress without admitting personal contribution.
- Assurance that the complaint will be investigated and a timeline of how long this will take and how the outcome will be communicated with any immediate action(s) that will be taken described.

When initially dealing with a complaint:

- Remain calm.
- Conduct the conversation in a private area or quiet zone.
- Thank the complainant for bringing the matter up and allow them to tell their story uninterrupted showing empathy and active listening.
- Make notes at the time or immediately afterwards.
- Try and clarify the resolution the complainant is seeking (it is dangerous to assume you know what the client wants as an outcome).
- Outline the 'what next'.

It may be clear from the letter or conversation that significant financial or legal redress is being sought, in which case the indemnity insurers should be informed.

When the complaint has been investigated then the complainant should be contacted with the outcome, any questions answered, and the proposed solution discussed hopefully reaching a satisfactory conclusion. If resolution is not possible then next steps for the complainant should be outlined which may be an internal appeals process, external mediation such as the Veterinary Client Mediation Service (UK), or escalation to the indemnity insurers or regulatory body.

Prevention

As with most situations, prevention is the best option with many complaints being avoided by good communication and recognising that a client is becoming upset or angry and dealing with it at that point.

If something has happened, such as a medical mistake, which you may feel will lead to a complaint then seek advice and guidance from colleagues, your indemnity insurers, or the regulator's professional conduct department.

In such circumstances, admitting to the client that something has happened rather than hoping they may not notice or actively trying to hide the mistake is essential professional conduct. Although unpleasant and uncomfortable, outcomes are almost invariably better and less stressful for all concerned.

Most issues that end up with disciplinary action by the regulator are not because of the mistake itself but actions taken after the mistake has occurred.

1.6.4 PROFESSIONAL CONDUCT

Professionalism is based on five key principles:

1. Professional competence.
2. Honesty and integrity.

3. Independence and impartiality.
4. Client confidentiality and trust.
5. Professional accountability.

A veterinary surgeon or veterinary nurse's professional responsibilities are usually outlined by the national body that oversees the professions. In the UK, the RCVS Code of Professional Conduct and supporting guidance provides this framework.

- Ensure animal health and welfare is their first consideration when attending to animals.
- Keep within their own area of competence and refer cases responsibly.
    - Competence is about having an appropriate level of skill, knowledge, support, and facilities to care for a patient, whether or not you have performed that procedure or surgery or managed a particular disease or condition before.
- Veterinary surgeons who prescribe, supply. and administer medicines must do so responsibly.
- Communicate with each other and with the client to ensure the health and welfare of animals under their care.
- Ensure that clinical governance forms part of professional activities.
- Be open and honest with clients and respect their needs and requirements.
- Provide independent and impartial advice and inform a client of any conflict of interest.
- Provide appropriate information to clients about the practice, including the costs of services and medicines.
- Ensure informed consent is obtained before treatments or procedures are carried out.
- Keep clear, accurate, and detailed clinical and client records.
- Do not disclose information about a client or the client's animals to a third party, unless the client gives permission, or animal welfare or the public interest may be compromised.
- Respond promptly, fully, and courteously to clients' complaints and criticism.
- Take reasonable steps to address adverse physical or mental health or performance that could impair fitness to practise; or, that results in harm, or a risk of harm, to animal health or welfare, public health, or the public interest.
- If you are concerned about a professional colleague's fitness to practise, take steps to ensure that animals are not put at risk and the public is protected.
- Maintain and develop knowledge and skills.
- Ensure that all their professional activities are covered by indemnity insurance.
- Work together and with others in the veterinary team and business, to coordinate the care of animals and the delivery of services.
- Ensure that tasks are delegated only to those who have the appropriate competence and registration.
- Show honesty and integrity when dealing with their professional regulator that may include disclosing cautions or convictions, physical limitations, etc.
- Certification of facts and signing letters or forms is a key duty afforded because of professional status, hence opinions must be honest, unbiased, and given with due care, taking into account the principles of certification, e.g.
  https://www.rcvs.org.uk/setting-standards/advice-and-guidance/code-of-professional-conduct-for-veterinary-surgeons/supporting-guidance/certification/.
- Activity or behaviour outside the clinic has an impact on the profession. If it brings the profession into disrepute or undermines public confidence then the professional regulator may become involved.

Most regulators will have a professional conduct department that are a huge resource and will provide advice and support.

1.6.5 CONTINUING PROFESSIONAL DEVELOPMENT

In most countries, continuing professional development (CPD) is mandatory to maintain registration. Exact requirements vary, as does the way that CPD should be recorded and quantified. In general, there is a move away from recording time spent to more reflective, outcome- and impact-based CPD.

For new graduates prescribed frameworks such as VetGDP are in place for early years development.

Wherever possible CPD should follow a plan with a goal of what is to be achieved and is part of an annual personal development plan.

Some CPD may be driven by the practice to introduce new systems, processes, guidelines, or quality improvement.

CPD can also be structured to achieve further professional qualifications.

What can be counted as CPD varies between countries and may require approval by the regulator in order to count towards the mandated requirement, e.g. Registry of Approved Continuing Education (RACE) programme in the USA.

What counts as CPD is often misunderstood and can be seen with a very clinical-based lens, e.g. a lecture on jaundice. CPD is much wider than this and all aspects of developing professional skills, knowledge, and competence can be CPD. CPD is personal so may vary widely between professionals depending on their role and career goals. If unsure, then many countries have an education function within the regulator that can be consulted.

1.6.6 PATIENT REFERRAL

At times owners may wish for care that requires more knowledge, skills, and facilities than can be provided by the attending clinician. Needing to refer is not an admission of poor competence or ability but an acknowledgement that no one can have the knowledge and skills to deal with every case.

Discussing referral is best initiated by the clinician as an option rather than a client demanding it. Referral can take a variety of forms:

- Internally within the practice or local group.
- To another general practice where there are professionals with specific skills.
- Single discipline referral centres.
- Multidisciplinary referral centres.

Many practices will have guidelines about referral, but it is part of professional conduct to ensure decisions as to referral options are centred around animal health and welfare.

When discussing options for referral it is important that the client is aware of the level of skills, knowledge, and facilities that a particular option affords along with the likely costs that may be involved and the time input that will be required.

If the owner decides to opt for referral, then good communication with the clinician/practice/centre to which the patient is being referred is essential to maximise benefit. Ideally this should include the reason for referral, the client's expectation, and a full set of notes, including laboratory tests, images, and reports.

Good referral should involve shared care between the referring clinician and the referral team to ensure best patient care and follow-up.

1.7 MENTAL HEALTH AND WELLBEING

1.7.1 *Maintaining your mental health and wellbeing* 1.7.2 *Looking out for others* 1.7.3 *Caregivers' burden*

INTRODUCTION

Mental health and wellbeing is a hugely important topic for veterinary professionals and can easily get forgotten amongst the daily demands of caring for patients and their owners. Paying attention to your mental health and wellbeing is part of your professional responsibilities.

1.7.1 MAINTAINING YOUR MENTAL HEALTH AND WELLBEING

Poor mental health and wellbeing is not uncommon in the veterinary professions and has a complex aetiology and myriad of causes amplified by being a caring career with compassion fatigue being an additional burden.

Actively engaging in your mental health and wellbeing not only has personal benefits but will also improve your patient care and outcomes.

Most practices and institutions have adopted a robust approach to supporting mental health and wellbeing and you should make sure you are familiar with the support (internal and external) that you can access.

Like most conditions early recognition and action improves outcomes. Knowing your own stressors and triggers, signs, and behaviours are key to recognising when your mental health and wellbeing is at risk. This is, however, somewhat passive, and an active approach to maintain/improve mental health and wellbeing will bring long term benefits.

1.7.2 LOOKING OUT FOR OTHERS

Everyone has a responsibility to look after the team around them. This does not mean that we all need to be mental health professionals, but it is about recognising signs of poor mental health and wellbeing in others.

There are no specific signs that are unique to poor mental health, so spotting that a colleague is struggling can be difficult but there are things we can look out for:

- Drop in functioning, e.g. not completing tasks, forgetting things.
- Problems in concentration, logical thought, and speech.
- Loss of desire to participate.
- Increased sensitivities.
- Decline in personal care.
- Mood swings.
- Changes in appetite.
- Unusual and odd behaviour.
- Suspicious and nervous behaviours.

If a colleague may be struggling with their mental health and wellbeing, the first step is about asking them how they are feeling and having time to listen to the reply. If you do not feel able so to do, then speak to someone else that you trust within the team/practice for support and guidance.

1.7.3 CAREGIVERS' BURDEN

Well established in the human field, this is an emerging area of study in veterinary medicine. What is clear is that looking after a sick pet especially where the disease is chronic and variable such as a diabetic patient or a pet going through chemotherapy places a significant caregiver burden on the owner that can impact their mental health and wellbeing. This impact

may be due to a whole variety or combination of factors such as delivering treatment, assessing their pet's status, time management, on-going costs of tests, and medication.

Recognising that caregiver burdens exist for our clients is an important first step and should be something that is considered when a plan for managing a condition is discussed. At follow-up appointments, asking not just about how they feel their pet is doing, but also how they are managing is vital to ensure best outcomes for the pet–owner combination. An owner should feel comfortable to discuss how they are managing as it may be entirely possible to change the plan, e.g. an owner who is struggling to give phenylpropanolamine three times daily to their pet may be able to manage a higher dose twice daily much more easily, significantly reducing the stress of having to rush home at lunch to medicate their pet.

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2

CLINICAL PRESENTATIONS

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2.1 SNEEZING AND NASAL DISCHARGE

Approach to sneezing and nasal discharge

Key aspects of history and physical examination

- Duration and progression of disease.
- Likelihood of infectious disease.
- Character of discharge: mucoid, serous, bloody (alone or mixed with secretions). Uni or bilateral discharge at the start.
- Halitosis.
- Facial pain, asymmetry, or ocular signs.
- Physical examination:
  - Assess for regional lymphadenopathy.
  - Percuss sinuses.
  - Tracheal pinch test.
  - Oral and aural examination.
  - Assess nasal airflow.

NOTES FOR FIGURE 2.1

- *Reverse sneezing = violent paroxysmal inspiratory effort, often confused by owners as retching, gagging, or coughing.
- †Occasionally non-nasopharyngeal disease can cause nasal discharge, e.g. pneumonia and oesophageal reflux. Uni or bilateral haemorrhage can occur with systemic hypertension or thrombocytopenia.
- §Bacterial culture of nasal discharge is usually positive, but primary bacterial infections are very rare.

2.2 COUGHING (FIGURE 2.2)

Approach to coughing

- Coughing should be differentiated from reverse sneezing, gagging, and retching.
- Consider recent exposure to allergens or irritants; seasonality; progression; single or paroxysmal coughs.
- *Many animals do not expectorate even when their cough is productive. Check whether there is a terminal retch/swallowing, which indicates that the cough is productive.
- Cardiac disease rarely causes coughing in cats.

2.3 DYSPNOEA (FIGURE 2.3)

Approach to dyspnoea

Key decisions

- Dyspnoea is caused by a failure of oxygenation of tissues (and removal of carbon dioxide) due to:
- Obstruction to airflow, e.g. tracheal collapse, feline asthma.
- Failure of diffusion across the alveolar membrane, e.g. pneumonia, pulmonary oedema.
- Failure of lung circulation, e.g. cardiac disease, pulmonary hypertension, pulmonary thromboembolism, anaemia.
- Deciding which of these processes is responsible for the dyspnoea significantly aids disease localisation.

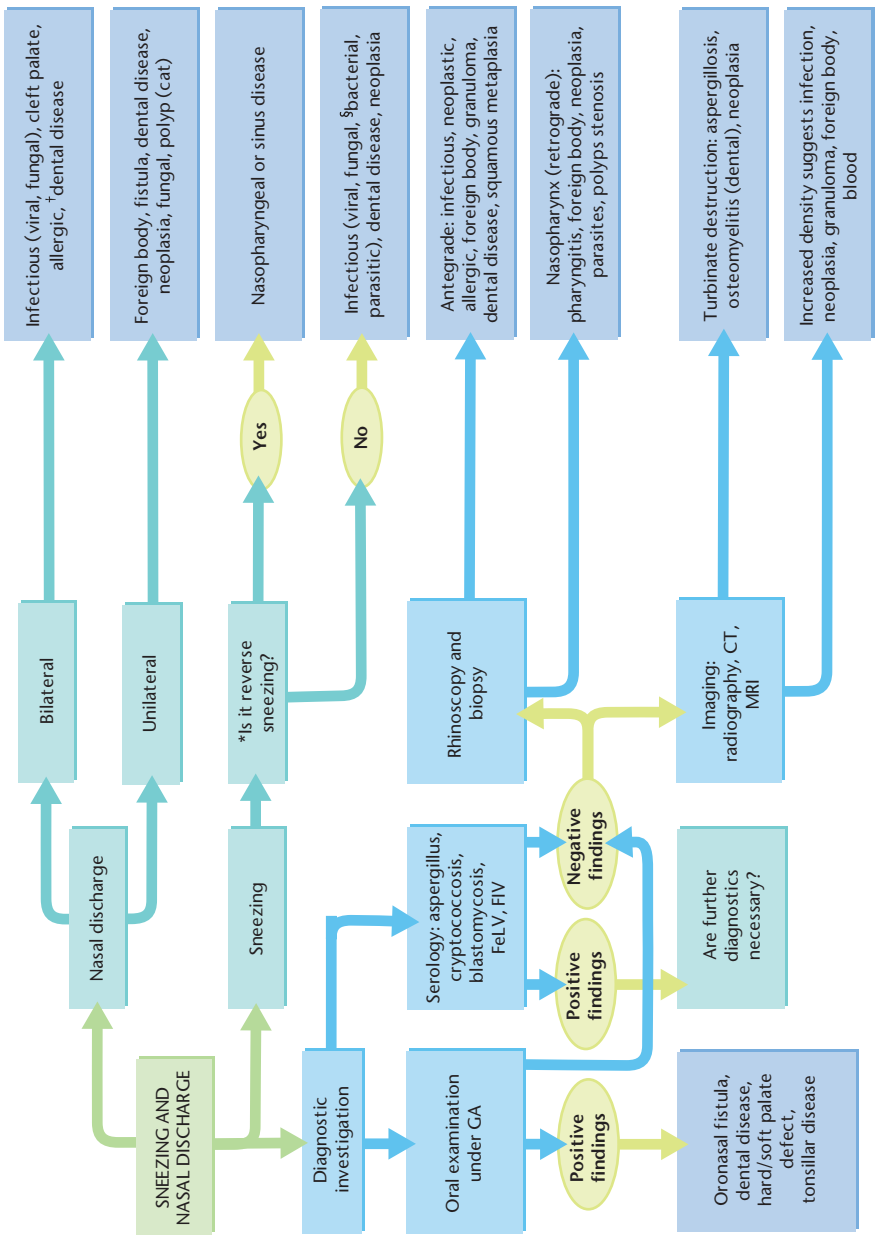

SNEEZING AND NASAL DISCHARGE
Nasal discharge
Bilateral
Unilateral
Sneezing
*Is it reverse sneezing?
Yes
No
Diagnostic investigation
Oral examination under GA
Serology: aspergillus, cryptococcosis, blastomycosis, FeLV, FIV
Rhinoscopy and biopsy
Positive findings
Negative findings
Positive findings
Are further diagnostics necessary?
Imaging: radiography, CT, MRI
Oronasal fistula, dental disease, hard/soft palate defect, tonsillar disease
Infectious (viral, fungal), cleft palate, allergic, †dental disease
Foreign body, fistula, dental disease, neoplasia, fungal, polyp (cat)
Nasopharyngeal or sinus disease
Infectious (viral, fungal, §bacterial, parasitic), dental disease, neoplasia
Antegrade: infectious, neoplastic, allergic, foreign body, granuloma, dental disease, squamous metaplasia
Nasopharynx (retrograde): pharyngitis, foreign body, neoplasia, parasites, polyps stenosis
Turbinate destruction: aspergillosis, osteomyelitis (dental), neoplasia
Increased density suggests infection, neoplasia, granuloma, foreign body, blood

Figure 2.1 Algorithm illustrating the differential diagnosis and diagnostic approach to sneezing and nasal discharge.

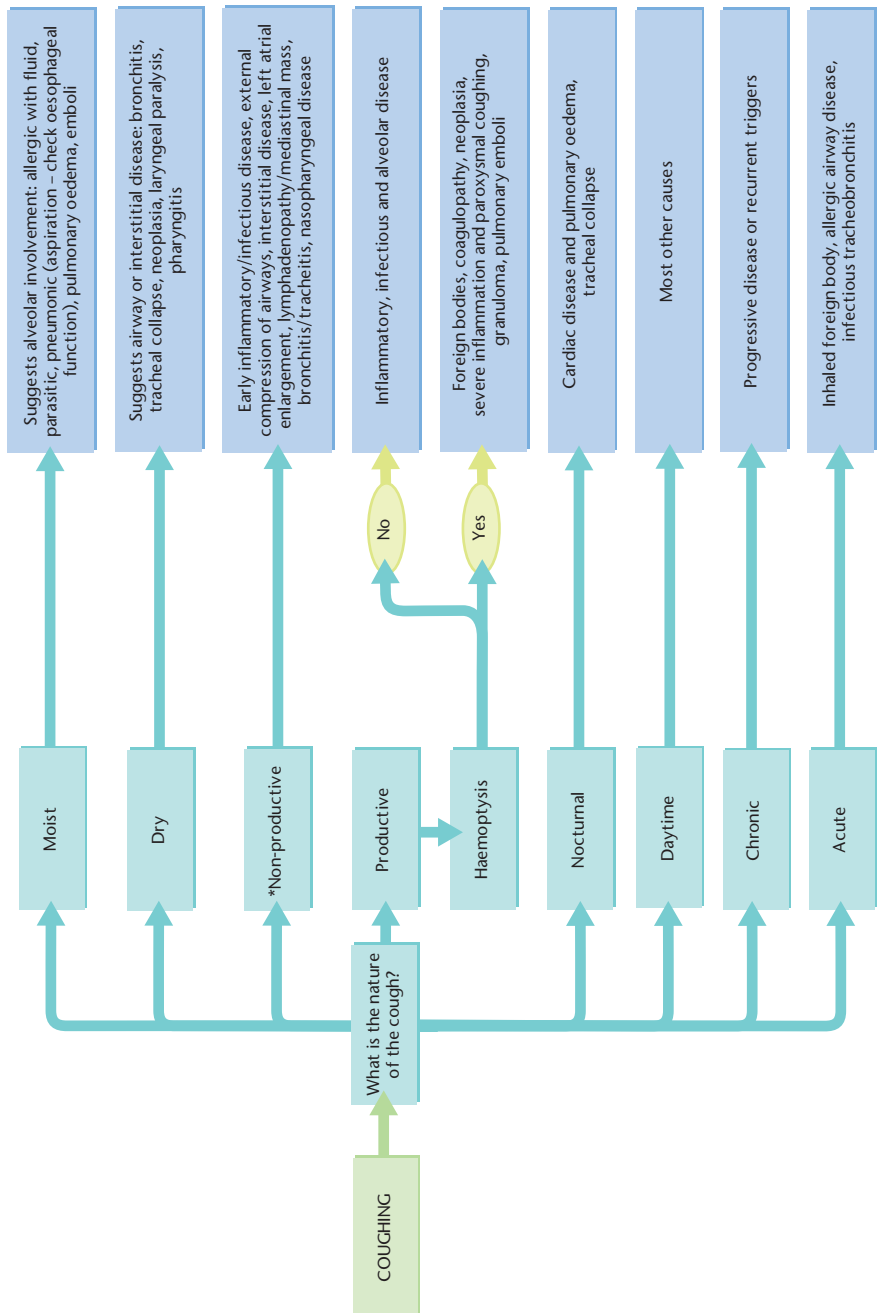

COUGHING
What is the nature of the cough?
Moist
Dry
*Non-productive
Productive
Haemoptysis
Nocturnal
Daytime
Chronic
Acute
No
Yes
Suggests alveolar involvement: allergic with fluid, parasitic, pneumonic (aspiration – check oesophageal function), pulmonary oedema, emboli
Suggests airway or interstitial disease: bronchitis, tracheal collapse, neoplasia, laryngeal paralysis, pharyngitis
Early inflammatory/infectious disease, external compression of airways, interstitial disease, left atrial enlargement, lymphadenopathy/mediastinal mass, bronchitis/tracheitis, nasopharyngeal disease
Inflammatory, infectious and alveolar disease
Foreign bodies, coagulopathy, neoplasia, severe inflammation and paroxysmal coughing, granuloma, pulmonary emboli
Cardiac disease and pulmonary oedema, tracheal collapse
Most other causes
Progressive disease or recurrent triggers
Inhaled foreign body, allergic airway disease, infectious tracheobronchitis

Figure 2.2 Flowchart illustrating likely cause of coughing based on clinical presentation.

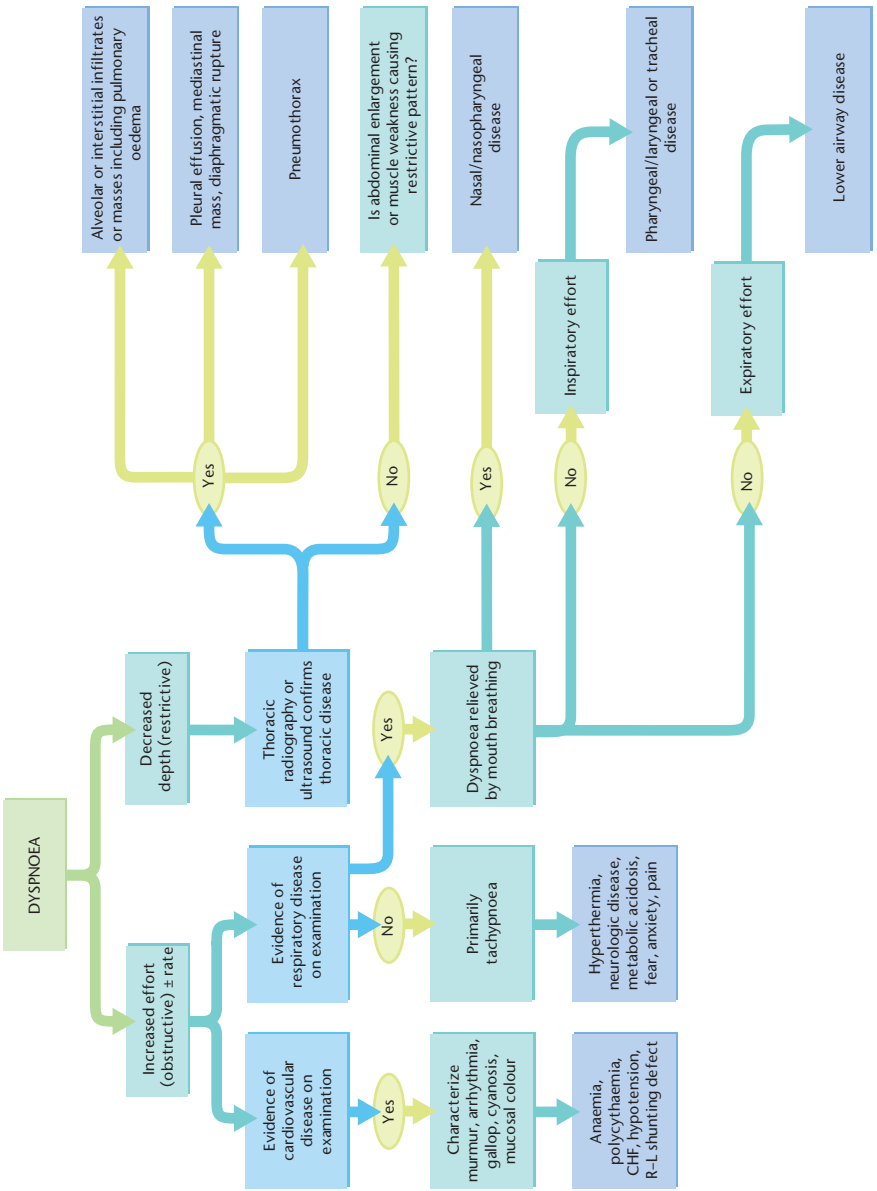


Figure 2.3 Algorithm illustrating an approach to dyspnoea based on presentation, clinical examination and initial investigation

Investigations

- Imaging single most valuable test in a dyspnoeic patient. Options ultrasound (echocardiography vs thoracic focused assessment with sonography for trauma, triage, and tracking [TFAST]), radiography, or CT.
- ***Alveolar/interstitial disease***: CBC, bronchoalveolar lavage with cytology, serology (fungal, heartworm [region dependent], lungworm [region dependent]), cardiac evaluation, bronchoscopy, guided biopsy, thoracotomy.
- ***Pleural disease***: CBC, biochemistry, FeLV, FIV, FIP, cytology and culture on sample, drainage, and repeat imaging, exploratory thoracotomy.

2.4 INAPPETENCE AND HYPOREXIA/ANOREXIA (FIGURE 2.4)

Approach to inappetence/anorexia

- Inappetence (pseudoanorexia)/anorexia is a very non-specific sign, particularly in some cats and small-breed dogs that normally have poor appetites, even when well.
- Inappetence is far more common than true anorexia and will usually resolve once the primary condition has been treated. Therefore, it is usually more productive to focus on the other clinical signs with which the patient presents.
- Underlying nausea is the most common cause of inappetence followed by pain associated with eating.
- Rarely, significant food aversion will have developed as a result of an underlying disease process resulting in persistent inappetence, potentially requiring long-term tube-feeding.

NOTE

*Manufacturers will sometimes change dietary formulation and this can cause problems for some pets. Was the inappetence associated with a new bag/batch of food, or a large bag of dry food that has spoiled?

2.5 VOMITING AND REGURGITATION (FIGURE 2.5)

Approach to vomiting and regurgitation

- Some animals will present with signs consistent with both vomiting and regurgitation.
- Vomiting and regurgitation is a common sequela to brachycephalic obstructive airway syndrome BUT these cases can have BOAS and primary GIT coexisting.
- The easiest way to decide whether material has been vomited (acidic) or regurgitated (alkaline) is to test the pH of the material using litmus paper. NB gastro-oesophageal reflux will be acidic.
- Chronic vomiting can lead to oesophagitis and secondary regurgitation associated with oesophageal dysfunction.

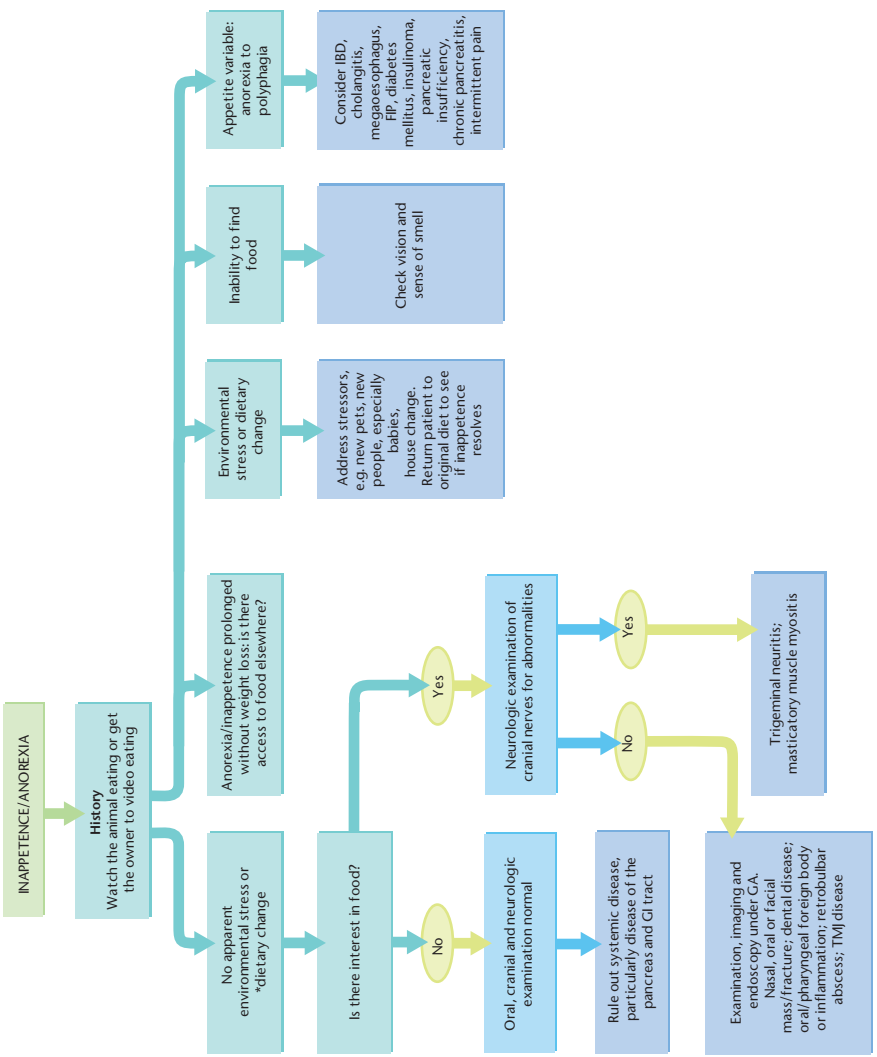

INAPPETENCE/ANOREXIA
History
Watch the animal eating or get the owner to video eating
No apparent environmental stress or *dietary change
Is there interest in food?
No
Oral, cranial and neurologic examination normal
Rule out systemic disease, particularly disease of the pancreas and GI tract
Yes
Neurologic examination of cranial nerves for abnormalities
No
Examination, imaging and endoscopy under G.A. Nasal, oral or facial mass/fracture; dental disease; oral/pharyngeal foreign body or inflammation; retrobulbar abscess; TMJ disease
Yes
Trigeminal neuritis; masticatory muscle myositis
Anorexia/inappetence prolonged without weight loss: is there access to food elsewhere?
Environmental stress or dietary change
Address stressors, e.g. new pets, new people, especially babies, house change. Return patient to original diet to see if inappetence resolves
Inability to find food
Check vision and sense of smell
Appetite variable: anorexia to polyphagia
Consider IBD, cholangitis, megaoesophagus, FIP, diabetes mellitus, insulinoma, pancreatic insufficiency, chronic pancreatitis, intermittent pain

Figure 2.4 Algorithm illustrating possible causes and diagnostic approach to inappetence and anorexia based on history and physical examination.

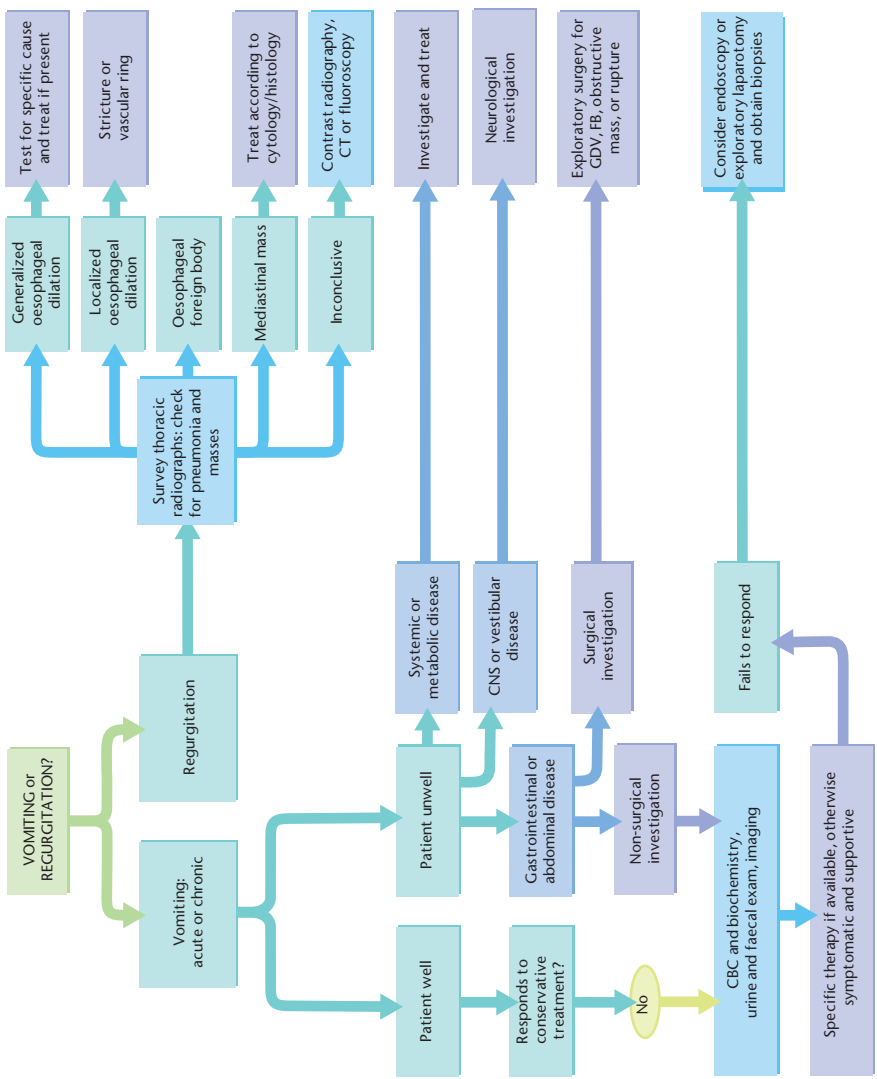

VOMITING or REGURGITATION?
Regurgitation
Vomiting: acute or chronic
Survey thoracic radiographs: check for pneumonia and masses
Generalized oesophageal dilation
Localized oesophageal dilation
Oesophageal foreign body
Mediastinal mass
Inconclusive
Test for specific cause and treat if present
Stricture or vascular ring
Treat according to cytology/histology
Contrast radiography, CT or fluoroscopy
Patient unwell
Patient well
Responds to conservative treatment?
No
Systemic or metabolic disease
CNS or vestibular disease
Gastrointestinal or abdominal disease
Surgical investigation
Non-surgical investigation
Investigate and treat
Neurological investigation
Exploratory surgery for GDV, FB, obstructive mass, or rupture
CBC and biochemistry, urine and faecal exam, imaging
Fails to respond
Consider endoscopy or exploratory laparotomy and obtain biopsies
Specific therapy if available, otherwise symptomatic and supportive

Figure 2.5 Algorithm illustrating an approach to patients with vomiting or regurgitation based on presentation, physical examination, and diagnostic results.

2.6 DIARRHOEA (FIGURE 2.6)

When presented with a patient with diarrhoea, an important first step is to decide whether the signs are marked i.e. acute severe diarrhoea but the patient is well from a patient whose signs may appear less marked but who is systemically unwell which would demand a more aggressive clinical approach.

2.7 WEIGHT LOSS (FIGURE 2.7)

NOTE

- Look for calorie loss through urine (protein/glucose) or due to severe skin disease.

2.8 POLYURIA/POLYDIPSIA (FIGURE 2.8)

As many cats go outside to urinate and may also drink outside, PU/PD can be relatively long standing in cats before the owner is aware or even go unnoticed.

2.9 FELINE IDIOPATHIC CYSTITIS (FIGURE 2.9)

The widespread use of antimicrobials is not indicated in cats with FIC; relatively few showing signs of cystitis have a significant bacterial infection although rates are higher in elderly female cats.

2.10 URINARY INCONTINENCE (FIGURE 2.10)

History is important in incontinent patients to understand when the incontinence occurs, how bad it is and whether the patient urinates normally most of the time.

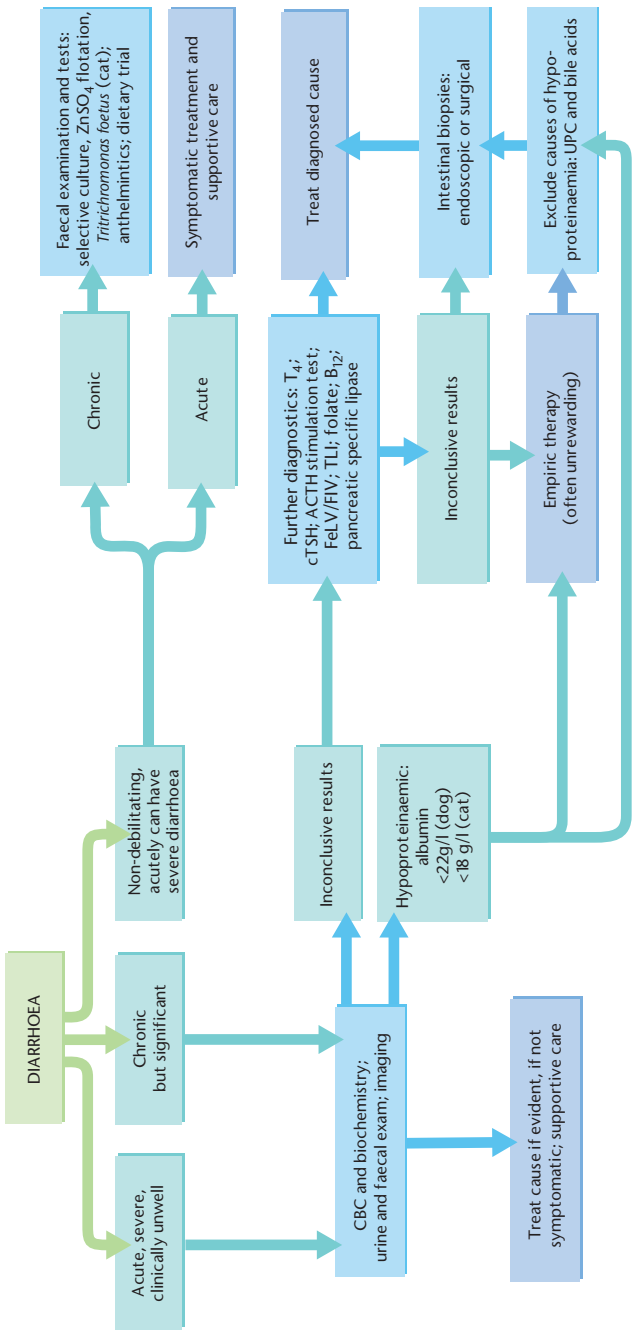

DIARRHOEA
Acute, severe, clinically unwell
Chronic but significant
Non-debilitating, acutely can have severe diarrhoea
CBC and biochemistry; urine and faecal exam; imaging
Inconclusive results
Hypoproteinaemic: albumin <22g/l (dog) <18 g/l (cat)
Treat cause if evident, if not symptomatic; supportive care
Chronic
Acute
Further diagnostics: T_4; cTSH; ACTH stimulation test; FeLV/FIV; TLI; folate; B_{12}; pancreatic specific lipase
Inconclusive results
Empiric therapy (often unrewarding)
Faecal examination and tests: selective culture, $ZnSO_4$ flotation, *Tritrichromonas foetus* (cat); anthelmintics; dietary trial
Symptomatic treatment and supportive care
Treat diagnosed cause
Intestinal biopsies: endoscopic or surgical
Exclude causes of hypo-proteinaemia: UPC and bile acids

Figure 2.6 Algorithm outlining the diagnostic approach to the diarrhoeic patient based on presentation.

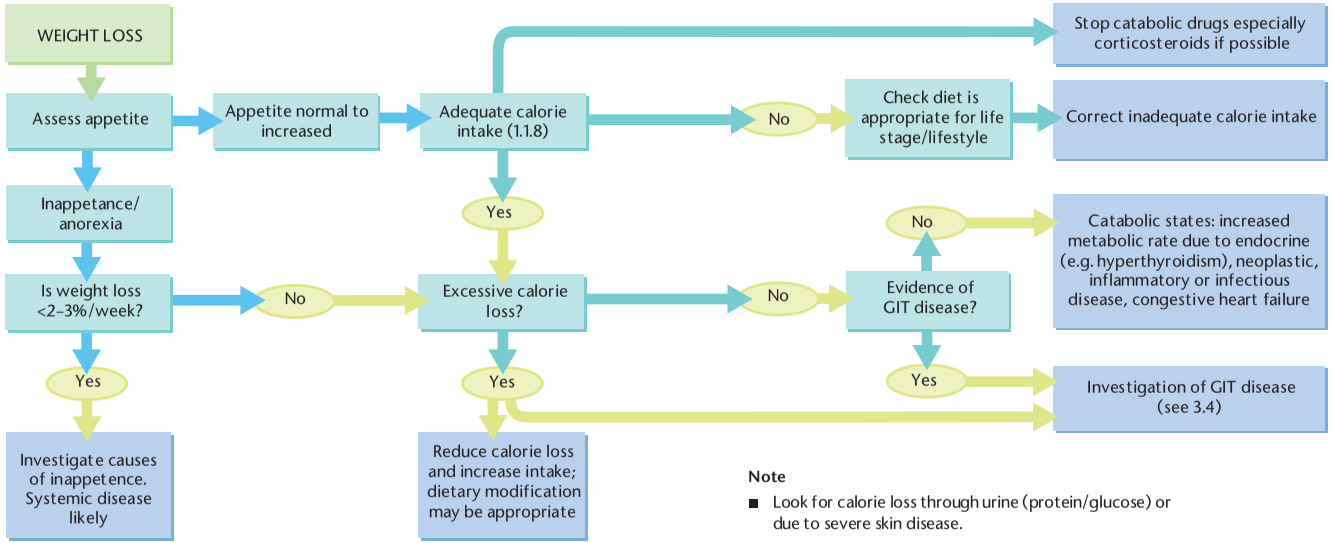

WEIGHT LOSS
Assess appetite
Appetite normal to increased
Adequate calorie intake (1.1.8)
No
Check diet is appropriate for life stage/lifestyle
Correct inadequate calorie intake
Stop catabolic drugs especially corticosteroids if possible
Inappetance/ anorexia
Yes
No
Catabolic states: increased metabolic rate due to endocrine (e.g. hyperthyroidism), neoplastic, inflammatory or infectious disease, congestive heart failure
Is weight loss <2–3%/week?
No
Excessive calorie loss?
No
Evidence of GIT disease?
Yes
Yes
Yes
Investigation of GIT disease (see 3.4)
Investigate causes of inappetence. Systemic disease likely
Reduce calorie loss and increase intake; dietary modification may be appropriate
Note
Look for calorie loss through urine (protein/glucose) or due to severe skin disease.

Figure 2.7 Algorithm outlining the diagnostic approach to weight loss based on appetite and key mechanisms leading to weight loss.

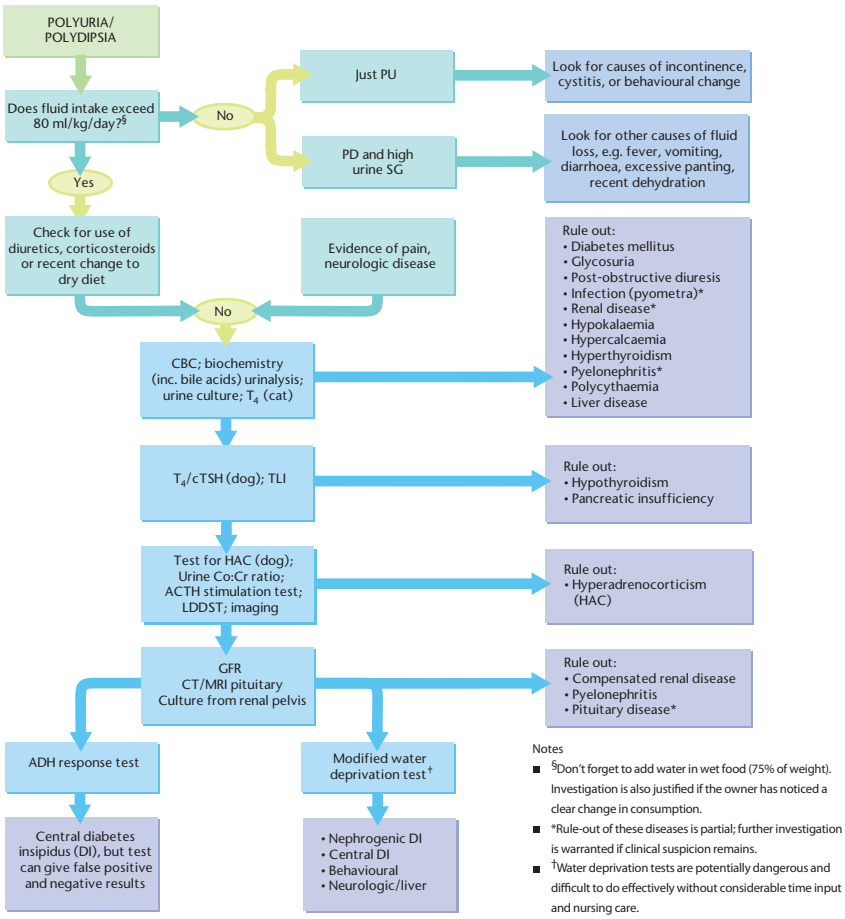

POLYURIA/ POLYDIPSIA
Does fluid intake exceed 80 ml/kg/day?§
No
Just PU
Look for causes of incontinence, cystitis, or behavioural change
PD and high urine SG
Look for other causes of fluid loss, e.g. fever, vomiting, diarrhoea, excessive panting, recent dehydration
Yes
Check for use of diuretics, corticosteroids or recent change to dry diet
Evidence of pain, neurologic disease
No
Rule out:
• Diabetes mellitus
• Glycosuria
• Post-obstructive diuresis
• Infection (pyometra)*
• Renal disease*
• Hypokalaemia
• Hypercalcaemia
• Hyperthyroidism
• Pyelonephritis*
• Polycythaemia
• Liver disease
CBC; biochemistry (inc. bile acids) urinalysis; urine culture; T4 (cat)
T4/cTSH (dog); TLI
Rule out:
• Hypothyroidism
• Pancreatic insufficiency
Test for HAC (dog); Urine Co:Cr ratio; ACTH stimulation test; LDDST; imaging
Rule out:
• Hyperadrenocorticism (HAC)
GFR
CT/MRI pituitary
Culture from renal pelvis
Rule out:
• Compensated renal disease
• Pyelonephritis
• Pituitary disease*
ADH response test
Modified water deprivation test†
Central diabetes insipidus (DI), but test can give false positive and negative results
• Nephrogenic DI
• Central DI
• Behavioural
• Neurologic/liver
Notes
§Don't forget to add water in wet food (75% of weight). Investigation is also justified if the owner has noticed a clear change in consumption.
*Rule-out of these diseases is partial; further investigation is warranted if clinical suspicion remains.
†Water deprivation tests are potentially dangerous and difficult to do effectively without considerable time input and nursing care.

Figure 2.8 Algorithm outlining the diagnostic approach based on fluid intake used to rule out the cause of PU/PD.

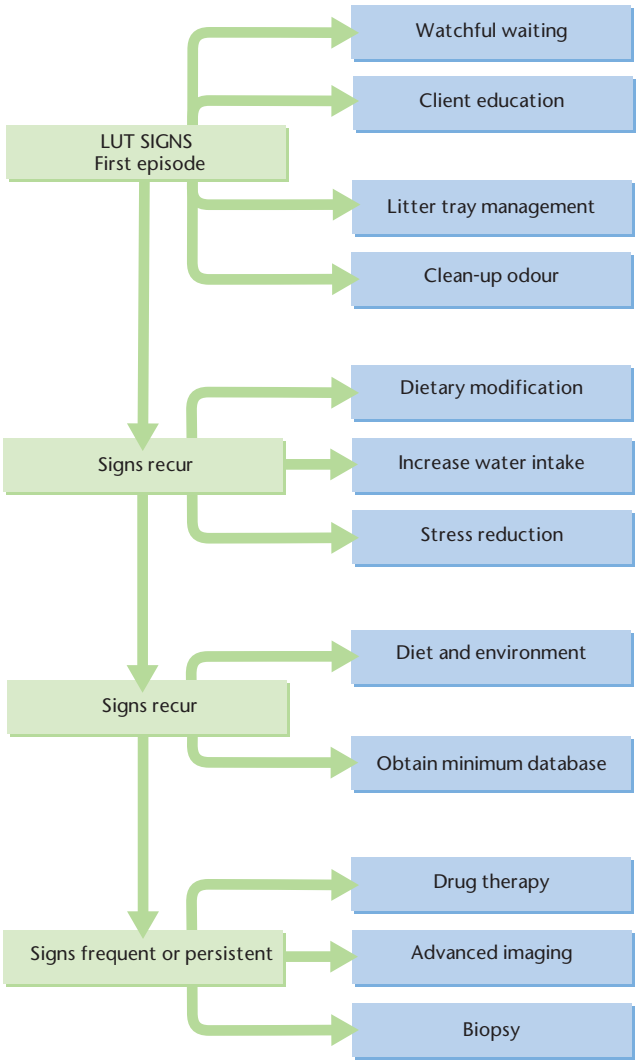
2.11 RENAL DISEASE (FIGURE 2.11)

Approach to renal disease

- Differentiating pre-renal vs acute kidney injury vs chronic kidney disease can be difficult. Acute and chronic changes may coexist, as animals with pre-existing renal disease are more at risk of suffering from an acute injury.
- The signs or changes in laboratory parameters listed are generally the case, but exceptions do occur.

NOTE

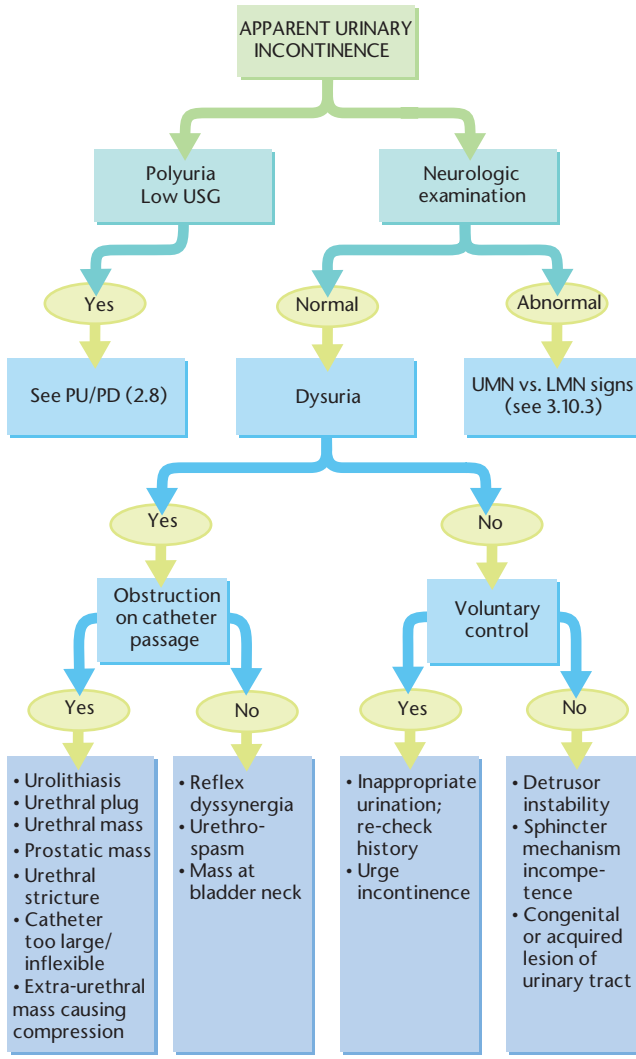
- *Suggests disease which interferes with ADH function, e.g. hypoadrenocorticism, hypercalcaemia, sepsis.



Approach to feline lower urinary tract disease (FLUTD)

- The majority (55–65%) of cases of FLUTD are idiopathic.
- These cases are best managed using a multimodal approach, involving changes to home environment, increasing water turnover through diet, and medication.

Figure 2.9 Algorithm outlining suggested approach to the investigation of FIC.



Note

- Be sure that is true incontinence; differentiate from inappropriae urination associated with behaviour, pollakiuria or PU/PD.

Figure 2.10 Algorithm outlining the approach to incontinence based on presentation and neurologic examination.

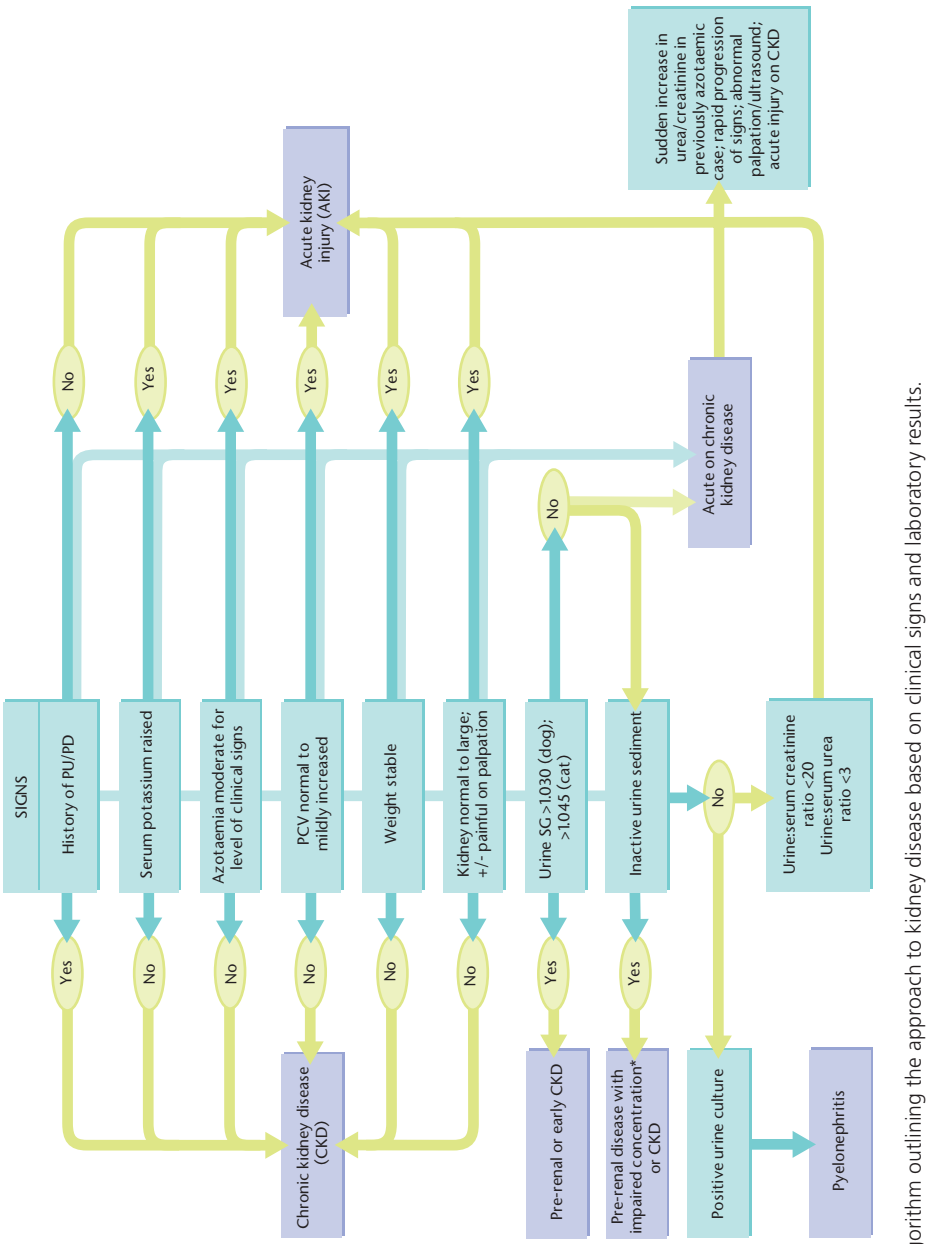

SIGNS
History of PU/PD
Serum potassium raised
Azotaemia moderate for level of clinical signs
PCV normal to mildly increased
Weight stable
Kidney normal to large; +/- painful on palpation
Urine SG >1.030 (dog); >1045 (cat)
Inactive urine sediment
Urine:serum creatinine ratio <20
Urine:serum urea ratio <3
Yes
No
Acute kidney injury (AKI)
Chronic kidney disease (CKD)
Acute on chronic kidney disease
Sudden increase in urea/creatinine in previously azotaemic case; rapid progression of signs; abnormal palpation/ultrasound; acute injury on CKD
Pre-renal or early CKD
Pre-renal disease with impaired concentration* or CKD
Positive urine culture
Pyelonephritis

Figure 2.11 Algorithm outlining the approach to kidney disease based on clinical signs and laboratory results.

2.12 PYREXIA (FIGURE 2.12)

Approach to pyrexia of unknown origin

- There is always pressure to treat a pyrexic case on presentation. However, watchful waiting and/or targeted investigation is frequently a better option.
- If the fever is high enough to require immediate treatment, use antipyretics rather than other treatments such as antimicrobials, which may interfere with subsequent investigation.
- Non-specific treatment with aspirin 10–25 mg/kg po q8–24 hr (dog); 10 mg/kg po q48–72 hr (cat); or paracetamol 10 mg/kg po q12 hr (dog). Other NSAIDs have variable antipyretic effects.
- Ranking of causes of pyrexia in referred cases: immune mediated disease > localised/systemic infection > neoplasia.
- Approximately 15% of referred cases are idiopathic

NOTES

- *Full haematology, biochemistry, urine, and faecal culture, FeLV/FIV.
- §Assuming no localising signs have developed, in which case investigation should be focused on these.
- †For tick-borne disease, coronavirus, toxoplasma, fungal disease (depending on location).
- ‡Informed owner consent is needed for trial with antimicrobials, corticosteroids, etc.

2.13 ANAEMIA (FIGURE 2.13)

Approach to anaemia/pallor

- Differential diagnosis of pallor:
  - Hypovolaemia.
  - Shock.
  - Poor cardiac output.
  - High sympathetic tone.
  - Anaemia.
  - Measure packed cell volume (PCV) to determine if anaemia is present and at a level to cause clinical signs: PCV <23% (dog); <18% (cat).

NOTES

- Anaemia can have multiple causes in a particular disease.
- Features of regeneration include:
  - Increased reticulocyte count.
  - High mean cell volume (MCV) (especially dogs).
  - Anisocytosis and polychromasia.

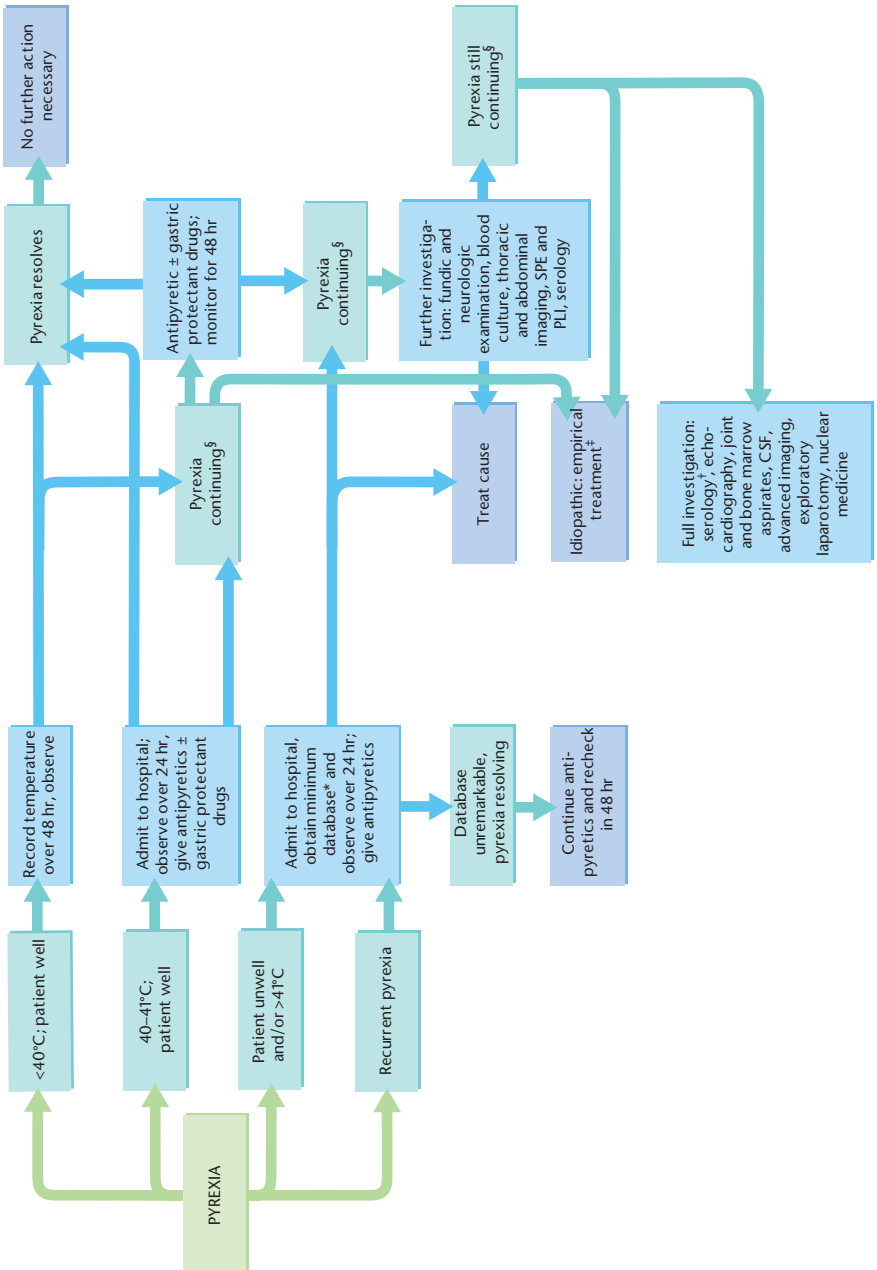

PYREXIA
<40°C; patient well
40–41°C; patient well
Patient unwell and/or >41°C
Recurrent pyrexia
Record temperature over 48 hr, observe
Admit to hospital; observe over 24 hr, give antipyretics ± gastric protectant drugs
Admit to hospital, obtain minimum database* and observe over 24 hr; give antipyretics
Database unremarkable, pyrexia resolving
Continue antipyretics and recheck in 48 hr
Pyrexia resolves
Antipyretic ± gastric protectant drugs; monitor for 48 hr
Pyrexia continuing§
Further investigation: fundic and neurologic examination, blood culture, thoracic and abdominal imaging, SPE and PLI, serology
Pyrexia still continuing§
No further action necessary
Pyrexia continuing§
Treat cause
Idiopathic: empirical treatment‡
Full investigation: serology†, echocardiography, joint and bone marrow aspirates, CSF, advanced imaging, exploratory laparotomy, nuclear medicine

Figure 2.12 Algorithm outlining the diagnostic approach to pyrexia according to temperature on presentation.

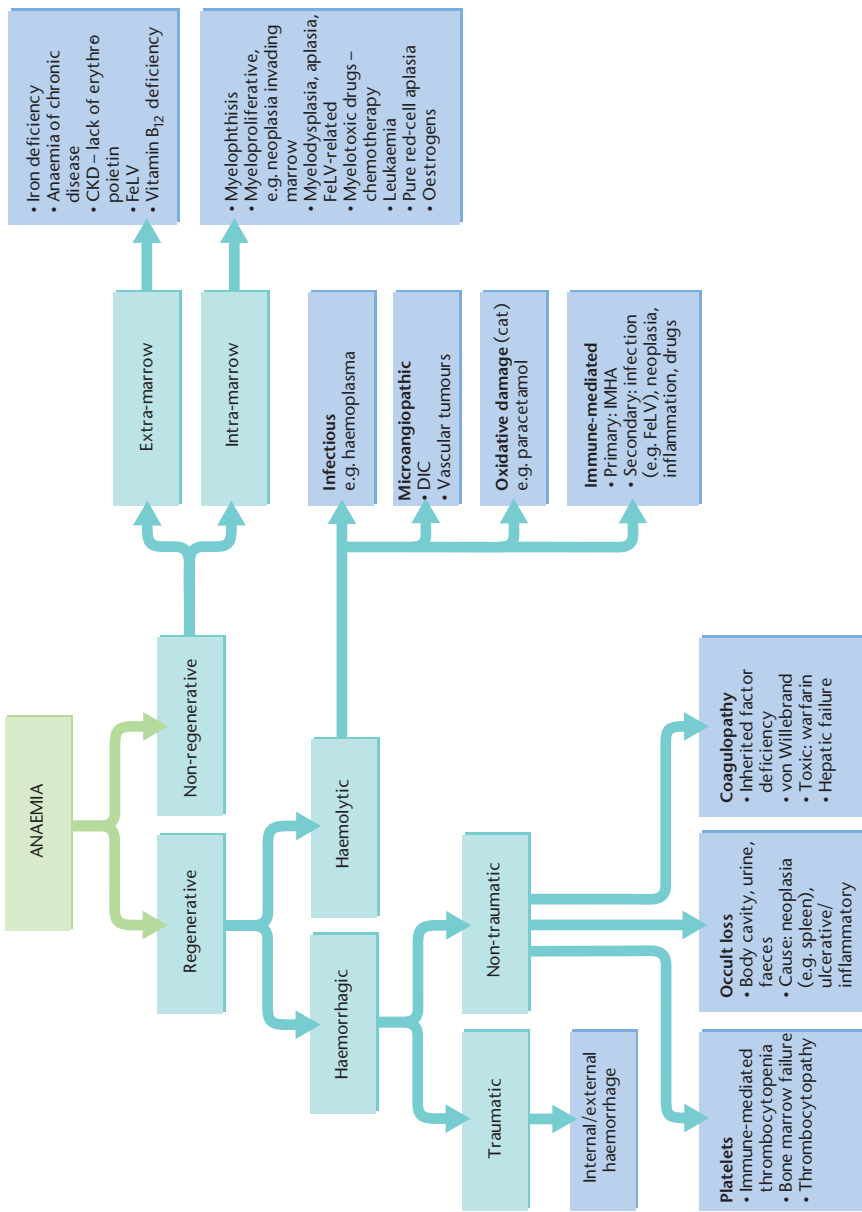


Figure 2.13 Algorithm outlining the diagnostic approach to anaemia based on the type of anaemia.

2.14 TREMOR (FIGURE 2.14 AND TABLE 2.1)

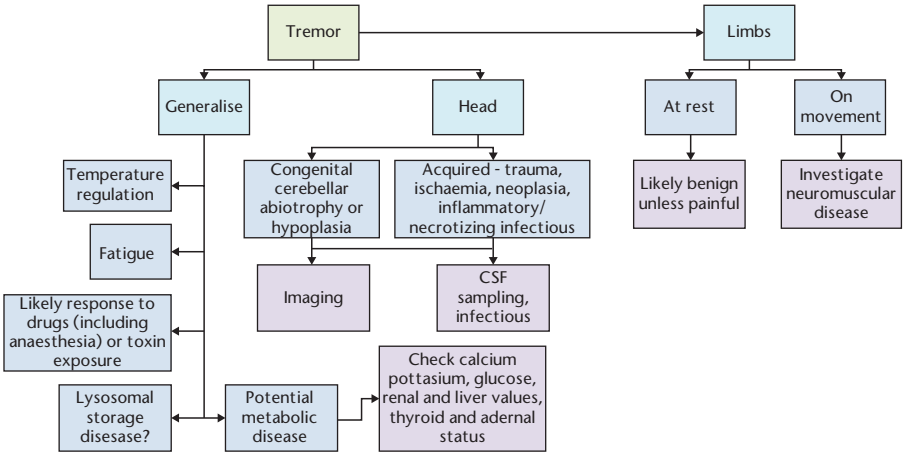


Figure 2.14 Algorithm outlining an approach to tremor based on location and history.

NOTES

- Describe tremor:
- *Shiver* – involuntary, high frequency, usually torso.
- *Tremble/quiver* – moderate frequency, coarse.
- *Intention* – exacerbated when attempt to coordinate movement, e.g. lowering head to a bowl to eat.
- *Postural* – more pronounced at rest.
- *Fasiculation* – rapid contraction/relaxation of adjacent muscle groups producing writhing sensation usually indicates nerve root irritation.
- *Myoclonus* – episodic jerking of a muscle group(s) may continue in sleep.

Table 2.1 Other disorders of muscle movement

Chorea	Abrubt, unsustained contraction of different muscle groups producing dance-like movements
Dystonia	Sustained, slow contraction of opposing muscles of a body region producing a twisted posture
Ballism	Abrybt contraction of the limb muscles which results in a flailing movement of the limb; often unilateral
Myokymia	Focal or generalized continuous muscle contraction, often exhibiting skin rippling overlying the affected muscle
Myotonus	Sustained contraction of a muscle group
Neuromyotonia	Continuous spontaneous muscle activity characterised by muscle rippling, muscle stiffness, and myotonia
Cramp	Sudden, severe, and involuntary muscle contraction or over-shortening that is generally temporary and benign
Tetanus	Sustained muscle contraction, usually the extensors, without relaxation
Tetany	Intermittent muscular contractions without relaxation
Paradoxical myotonia	Delayed muscle relaxation that occurs and worsens with exercise and improved with rest

- Isometric pelvic limb tremor common in older, small-breed dogs, occurs during standing, is benign and disappears during voluntary movement.
- Partial seizure can be mistaken for tremor.

2.15 ATAXIA (FIGURE 2.15)

NOTES

- Ataxia, paresis, and paralysis associated with disease of brain, spine, and peripheral nerves.
- * Vestibular ataxia usually easiest to recognise, may be accompanied by nystagmus. Side of the head tilt usually denotes side of lesion.
- Other non-neurologic conditions are often described as 'ataxia' and these need to be recognised as an expression of weakness, disorientation, pain, intoxication, etc.

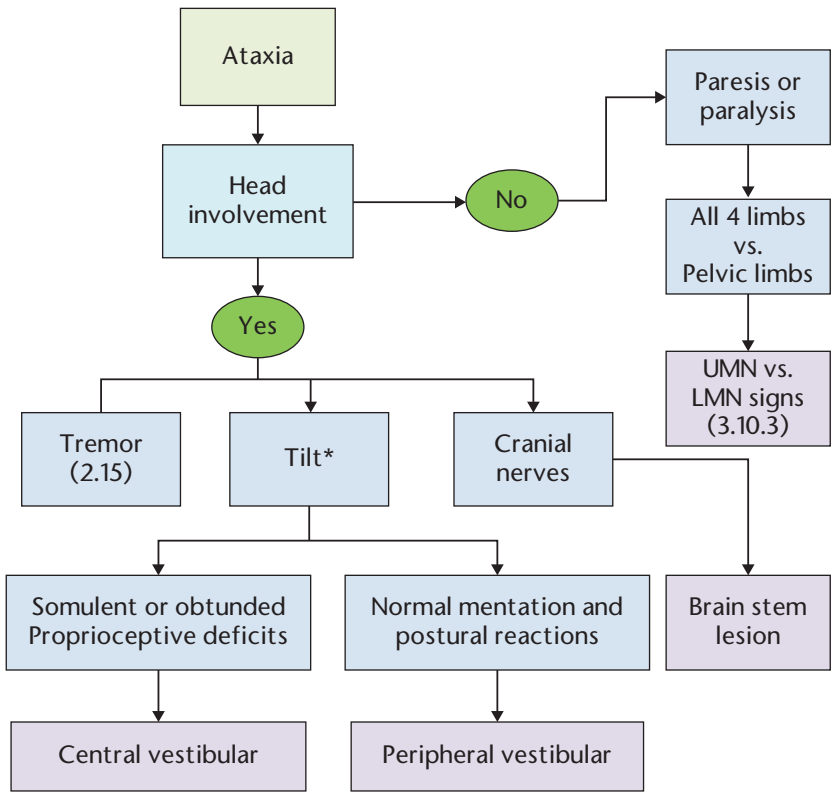


Figure 2.15 Algorithm outlining an approach to ataxia based on location and neurologic examination.

2.16 JAUNDICE (FIGURE 2.16)

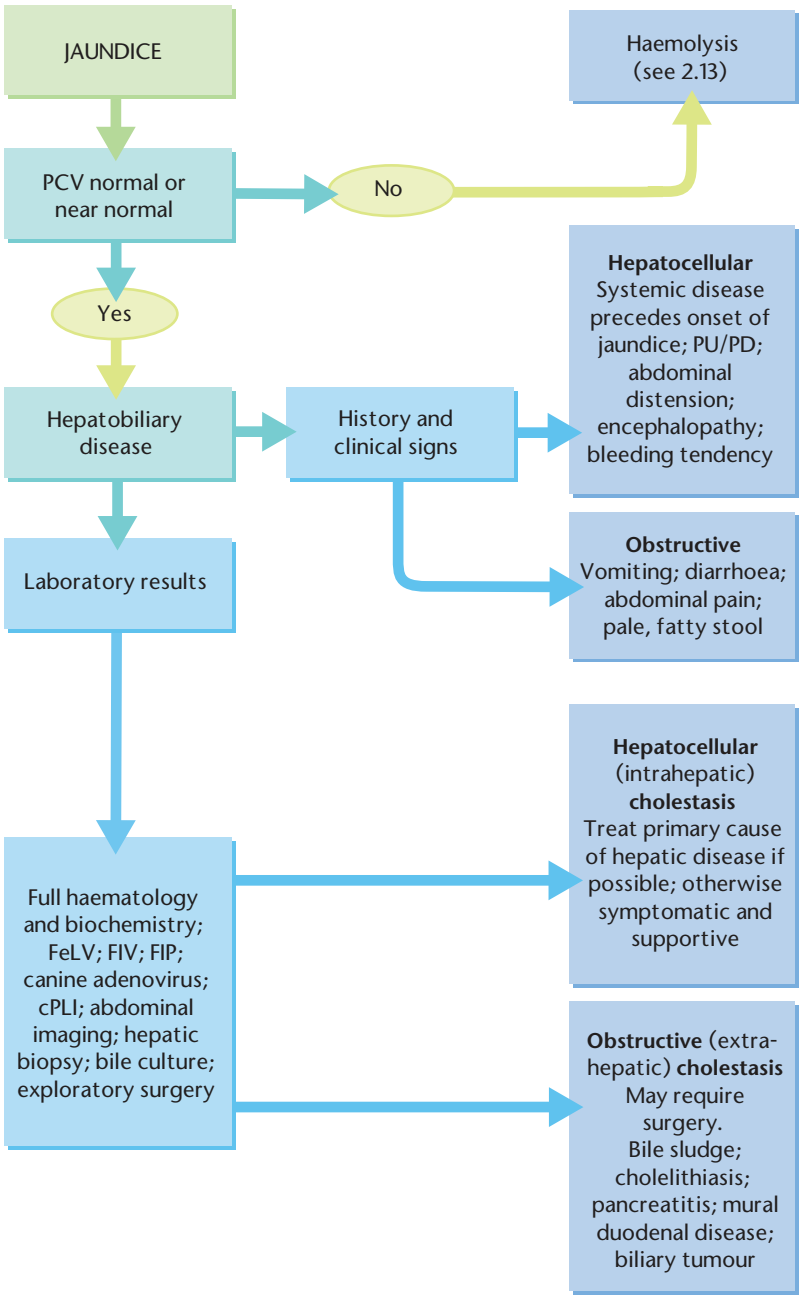

JAUNDICE
PCV normal or near normal
No
Haemolysis (see 2.13)
Yes
Hepatobiliary disease
History and clinical signs
Hepatocellular Systemic disease precedes onset of jaundice; PU/PD; abdominal distension; encephalopathy; bleeding tendency
Obstructive Vomiting; diarrhoea; abdominal pain; pale, fatty stool
Laboratory results
Full haematology and biochemistry; FeLV; FIV; FIP; canine adenovirus; cPLI; abdominal imaging; hepatic biopsy; bile culture; exploratory surgery
Hepatocellular (intrahepatic) cholestasis Treat primary cause of hepatic disease if possible; otherwise symptomatic and supportive
Obstructive (extra-hepatic) cholestasis May require surgery. Bile sludge; cholelithiasis; pancreatitis; mural duodenal disease; biliary tumour

Figure 2.16 Algorithm outlining an approach to jaundice based on history, clinical signs, and laboratory results.

NOTES

- Abdominal imaging should include detailed ultrasound of the liver, gall bladder, extrahepatic bile duct, duodenal papilla, and pancreas.
- Bile acids are unreliable in jaundiced patients.
- Cats with extrahepatic biliary obstruction do not always have dilation of the gall bladder.
- FIP can cause jaundice without clear hepatic involvement.
- Starvation can lead to jaundice especially in cats.

3

BODY SYSTEMS AND MULTISYSTEMIC DISEASE

DOI: 10.1201/9780429186783-3

3.1 EYES

3.1.1 *Approach to diseases of the eye* 3.1.2 *Ophthalmic examination* 3.1.3 *Diagnostic tests and objectives* 3.1.4 *Topical ocular products* 3.1.5 *Role of nutrition* 3.1.6 *Diseases by anatomic area* 3.1.7 *Glaucoma*

INTRODUCTION

3.1.1 APPROACH TO DISEASES OF THE EYE

Diseases of the eye are best evaluated by deciding which anatomic structures within the eye are affected.

Many systemic, especially infectious, diseases can present with ocular signs, e.g. uveitis as a result of FIP; or retinal haemorrhage associated with hypertension.

HISTORY AND PHYSICAL EXAMINATION

3.1.2 OPHTHALMIC EXAMINATION

- Technique
  - Use a quiet, well-illuminated room which can be darkened.
  - Does the patient appear visual?
  - Is there ocular discharge present – uni or bilateral, describe?
  - Is there symmetry of the ocular structures such as exophthalmus, anisochoria?
  - Use bright focal light to examine the adnexa, anterior chamber, and PLR. Magnification is preferable.
  - Dilate the pupil (mydriasis – use 1% tropicamide and allow 20 minutes) to allow fundic examination.
- Direct ophthalmoscopy (Figure 3.1a and b)
  - Distant direct – set at 0 or $^{+}1$ looking for black opacities on the reflected path from the tapetum.
  - Close direct – set at $^{-}2$ to $^{+}2$; find and evaluate the optic disc and then the rest of the fundus. Then focus back through the anterior segment (lens about $^{+}10$).
- Indirect ophthalmoscopy

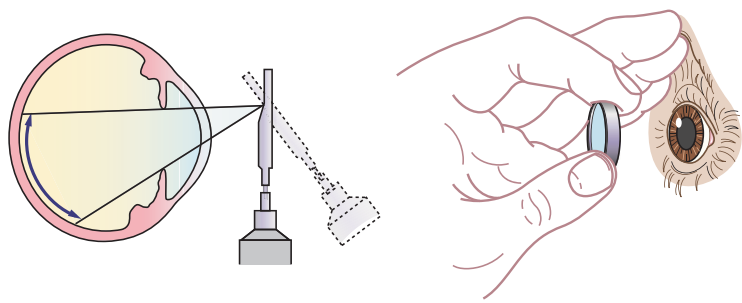


Figure 3.1 (a) **Direct ophthalmoscopy**. The ophthalmoscope is held close to the cornea and the angle adjusted to fully examine the retina. (b) **Indirect ophthalmoscopy**. The upper lid is held open, the lens is aligned and the hand and lens move with the head.

In its simplest form, requires a light and hand lens (Figure 3.2). Start with the lens close to the eye and withdraw until the image fills the field of view. Keep the lens at 90° to the light beam.

DIAGNOSIS

3.1.3 DIAGNOSTIC TESTS AND OBJECTIVES

- ***Menace response***: is the patient visual?
- ***Dazzle reflex and pupillary light responses***: part of the neuro-ophthalmic examination (pXX).
- ***Schirmer tear testing***: place the short end of the tip in lateral half of lower conjunctival sac holding the eyelid closed.
    - Measure over 1 minute. Dogs should produce 15–25 mm, but breed variations do occur. Cats normally over 9 mm.
- ***Fluorescein*** demonstrates ulceration and nasolacrimal patency.
    - Note that deep ulcers do not stain centrally; mucus on the corneal surface can give a 'false positive' so flush excess fluorescein away with saline and use a cobalt blue filter in a dark room to examine cornea.
- ***Tonometry*** is increasingly part of routine examination; reference interval for intraocular pressure (IOP) 12–25 mmHg for cats and dogs
- ***Blood tests*** primarily serve to look for systemic conditions causing ocular signs.
- ***Cytology and histology*** can provide valuable information in diseases affecting the eyelid, conjunctiva, and cornea.
- ***Infectious disease***: aerobic bacterial culture is appropriate for the majority of suspected infections. Special transport media are required for *Chlamydophila* and *Mycoplasma*. Alternative is to send a dry swab for infectious disease PCR in cats.
    - Fungal disease should be considered in animals that have travelled abroad but indigenous cases may increase as climate changes.
- ***Imaging***: ultrasound, CT, and MRI are valuable for investigating disease of the deeper structures of the eye, retrobulbar area, and optic nerve.
    - Radiographs have limited value, except to detect radiopaque foreign bodies and diseases affecting the orbital bone.
- ***Specialist techniques***: electroretinography to evaluate retinal function; gonioscopy to evaluate drainage angle.

TREATMENT

3.1.4 TOPICAL OCULAR PRODUCTS

- Provided the patient is amenable, topical therapy (see Table 3.1a) is the route of choice for diseases of the anterior segment of the eye, as it maximises exposure to the drug while minimising systemic side-effects. For posterior segment disease systemic therapy is required.

LACRIMAL FLUSHING

- In many cases, lacrimal flushing via the upper (dorsal) punctum can be undertaken with the patient conscious or sedated rather than anaesthetised. Fine nasal cannulae (23–24 g) are needed with topical local anaesthetic.

NUTRITION

3.1.5 ROLE OF NUTRITION

- Nutrition does not play a crucial role in the genesis or management of ocular disease. Notable exceptions would include taurine deficiency, vitamin E deficient retinopathy, and lipaemia retinalis.

Table 3.1a Topical ocular products	
Topical antimicrobials	*Licensed (UK)* **– Cloxacillin – penicillin mainly Gram positive (ointment), fucidic acid – mainly Gram positive (ointment), gentamicin – aminoglycoside mainly Gram negative (gel and drops)** *Unlicensed commonly used* **– chloramphenicol (drops and ointment) – broad spectrum, ofloxacin (drops) broad spectrum including** *Pseudomonas* **pp.**
Antivirals	Used in the management of FHV-1 keratitis Ganciclovir (ointment), famciclovir (oral), cidofovir (topical and injectable, not UK)
Glucocorticoids	Formulation affects depth of penetration Contraindicated – corneal ulceration
NSAIDs	Pain relief and management of uveitis
Ciclosporin	Immune-mediated keratoconjunctivitis sicca; chronic superficial keratoconjunctivitis
Glaucoma treatment	Carbonic anhydrase inhibitors – dorzolamide Prostaglandin analogues – latanoprost Parasympathomimetics – pilocarpine β-adrenergic blockers* – timolol maleate
Mydriatics	Dilate pupil for examination – tropicamide (rapid onset, shorter duration), surgery, and in anterior uveitis – atropine (longer duration)
Tear substitutes	Aqueous/mucin replacing – sodium hyaluronate and carbomer gels used most commonly Lipid replacing
Local anaesthetic	Proxymetacaine – rapid acting allows minor diagnostic and surgical procedures. Should not be used therapeutically

* use with care in patients with hypotension or cardiovascular disease.

- Lysine has been recommended as adjunctive treatment for chronic FHV-1; efficacy has not been shown.

SELECTED TOPICS

3.1.6 DISEASES BY ANATOMIC AREA

- See Table 3.1b. Note that injury/trauma, foreign bodies, and neoplasia can affect all areas.

3.1.7 GLAUCOMA

- ***Signs*** include:
  - Acute: pain, corneal oedema or scleral injection, unresponsive pupil, episcleral congestion, vision loss.
  - Chronic: globe enlargement, corneal ulceration and neovascularisation, scleral thinning, iris atrophy, lens luxation, cataract, haemorrhage, blindness.
- Glaucoma is classified as primary in a number of dog breeds, or secondary to other ocular pathology, such as lens luxation, uveitis, intraocular haemorrhage, ocular melanosis, or swollen lens, e.g. in diabetes.
- ***Therapy***: lower the intraocular pressure to <20 mmHg using surgical or medical therapy, e.g. osmotic diuretics, carbonic anhydrase inhibitors, miotics, β-adrenergic blockers.

Table 3.1b Ocular diseases by anatomic area

Area	Defect	Causes
Eyelid and nictitating membrane	Congenital	Coloboma, epibulbar dermoid, punctal atresia
	Eyelid position	Entropion (can be breed-related), ectropion, e.g. diamond eye in Clumber spaniels
	Ptosis	Defect CN III or Horner's syndrome
	Blepharitis	Infection/inflammation of eyelid
	Distichiasis	Ectopic cilia, trichiasis (facial hair on ocular surface) e.g. nasal fold in brachycephalic dogs
	Eosinophilic plaque (cat)	
	3rd eyelid	Scrolling, prolapse, cherry eye, inflammation, protrusion (suggests underlying disease), neoplasia, diarrhoea associated (cats)
Conjunctiva	Primary	Viral, irritant, allergic, immune-mediated FHV-1, FCV (cat) *Chlamydophila felis*, mycoplasma
	Secondary	Bacterial, mechanical
	Symblepharon – adhesion to itself/cornea, common in cats	
Cornea	Congenital	Dermoid, opacity, microcornea
	Dystrophy – thinning of the cornea or lipid/mineral dystrophy	
	Keratitis	Often ulcerative associated with eyelid abnormalities, immune-mediated, tear film, infection
	Pannus (chronic superficial keratoconjunctivitis) Herpetic keratitis (cat)	
	Exposure – loss or incomplete blink	CNV, anaesthesia
	Corneal sequestrum (cats)	
	Eosinophilic (proliferative) keratoconjunctivitis (cats)	
Uveal tract	Developmental	Deficiency in pigmentation, coloboma and uveal cysts, persistence of embryonic pupillary membrane
	Benign melanosis (dog) – foci of pigment	
	Synechiae: adhesions iris to cornea or lens	
	Uveitis: pain, photophobia, lacrimation	Infectious, e.g. FIP, parasitic, traumatic, immune mediated, lens-induced, intraocular foreign body
	Neoplasia	Melanoma, lymphoma
	Lipaemic aqueous (check serum triglycerides)	

Area	Defect	Causes
Lens	Congenital	Missing/small, coloboma, embryonic remnants
	Nuclear sclerosis (age-related)	
	Cataract	Primary – hereditary most common cause Secondary to other ocular disease, trauma, diabetes, hypocalcaemia, toxic, senile
	Luxation	Inherited (terriers), glaucoma, cataract, uveitis
Retina*	Normal	Significant variation
	Heritable disease	Collie eye anomaly, retinal dysplasia
	Acquired disease	Generalized progressive retinal atrophy, sudden acquired retinal degeneration, retinal detachment, lipaemia, taurine deficiency (cat) chorioretinitis (often indicates systemic disease, e.g. FIP, *Toxoplasma*, *Ehrlichia*, *Aspergillus*)

3.2 TEETH AND GUMS

3.2.1 *Introduction* 3.2.2 *Dental examination* 3.2.3 *Diagnostic tests* 3.2.4 *Dental score charts* 3.2.5 *Staging periodontal disease* 3.2.6 *Management of dental disease* 3.2.7 *Tooth extraction* 3.2.8 *Nutrition and dental disease* 3.2.9 *Equipment*

3.2.1 INTRODUCTION

- Dental disease is the most common pathology found on physical examination of cats and dogs.
- Association has been made between chronic inflammatory diseases including a number of dental pathologies, and systemic diseases such as renal disease.

HISTORY AND PHYSICAL EXAMINATION

3.2.2 DENTAL EXAMINATION

- The following areas should all be examined:
  - Oropharynx: palate, tonsils, caudal oral mucosa.
  - Lips and cheeks: mucocutaneous junction, philtrum, frenula, salivary papilla.
  - Oral mucosa: buccal, lingual, gingival surfaces.
  - Hard palate.
  - Floor of mouth and tongue, lingual frenulum.
  - Teeth (anatomy Figure 3.2a): life stage, missing/extra teeth, abnormalities of individual teeth, wear patterns, pathological changes, e.g. traumatic injuries such as fractures and tooth discolouration and lesions such as dental caries.
- Full oral examination requires the following:
  - Time to record findings using a standard dental chart, so that severity and change over time may be assessed.

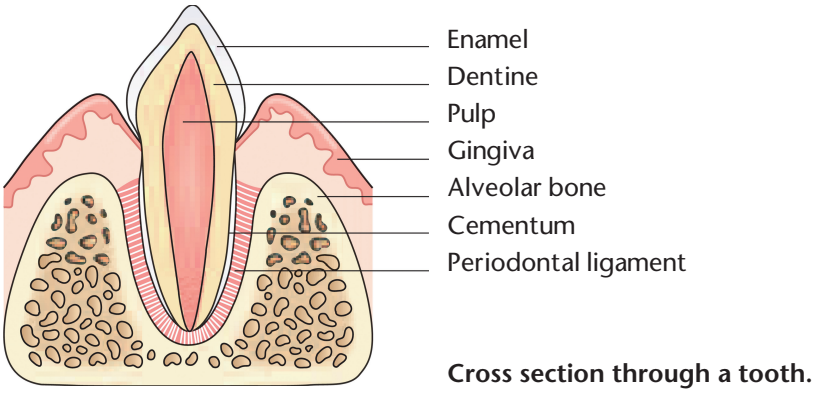


Figure 3.2a Cross-section through a tooth.

- General anaesthesia is required in order to carry out periodontal probing and dental radiography.

✪ Screening blood tests are often recommended prior to general anaesthesia, to assess for systemic diseases that may affect anaesthetic protocols. There should be an agreed action plan of response should abnormalities be found.

DIAGNOSIS

3.2.3 DIAGNOSTIC TESTS

- Thorough visual examination and periodontal probing in good lighting, together with dental radiographs, are essential diagnostic tests for all dental cases.
- Skull radiography is not recommended to assess dental disease as interpretation is made very difficult by superimposition of orodental structures. Full mouth radiography is recommended, using dental specific digital radiography (DR) or computed radiography (CR) image-capturing systems along with a dental x-ray generator
- Viral screening (FIV, feline calicivirus (FCV)) can be considered in cats but is unlikely to give a prognostic indicator for the progression of disease.

3.2.4 DENTAL SCORE CHARTS

- On a dental record card, record evidence of:
  - Gingivitis.
  - Periodontal probing depth.
  - Gingival recession.
  - Periodontal index.
  - Loss of tissue between multi-rooted teeth (furcation exposure).
  - Tooth mobility.
  - Degree of loss of tooth attachment.
  - Traumatic injuries such as fractures or discoloured teeth.
  - Oral mucosal inflammatory conditions such as feline chronic gingivostomatitis.
  - Visible oral tooth resorption lesions.
  - Any abnormal gingival enlargement.
  - Any other orodental lesion.

3.2.5 STAGING PERIODONTAL DISEASE

- Periodontal disease results from plaque-induced inflammation.
- The vast majority of cats and dogs over 3 years of age have a degree of disease which warrants intervention.
- The earliest stage of periodontal disease, gingivitis, is reversible.
- Once there is bone loss, changes become irreversible. The stages are defined as:
  - ***Gingivitis***: inflammation limited to gingiva.
  - ***Gingival enlargement***: increased periodontal probe depth (a pseudopocket) but no loss of periodontal support.
  - ***Periodontitis with vertical bone loss***: early loss of periodontal support and destruction of alveolar bone, root not yet exposed.
  - ***Periodontitis with horizontal bone loss***: loss of periodontal ligament and exposure of the root.

TREATMENT

3.2.6 MANAGEMENT OF PERIODONTAL AND DENTAL DISEASE

- The most important element is the maintenance of oral hygiene – tooth brushing (q24 hr with toothpaste). Dental diets and dental chews and oral antiseptics (topical chlorhexidine) can also be considered.
- Cats and dogs can develop an abnormally severe mucosal inflammatory response called feline chronic gingivostomatitis (cat) and chronic ulcerative paradontal stomatitis (dogs). These conditions require meticulous dental hygiene and where this is not possible, elective dental extractions may be considered to reduce the oral inflammatory stimulus. Immunosuppressive medications may also be considered for cases that are refractory to elective dental extractions.
- Therapy for periodontal disease includes:
  - Supragingival scaling and polishing.
  - Subgingival scaling, root planing, and polishing.
  - Sulcular lavage.
  - Periodontal surgery: gingivoplasty, tooth removal.

3.2.7 TOOTH EXTRACTION

- ***Indications***: advanced periodontitis, advanced destruction of dental hard tissue including tooth resorption, persistent primary teeth, malocclusion, traumatic tooth injury, overcrowding.
- ***Techniques***: be sure to use sharp, well-maintained instruments of the correct size.
- ***Closed***: extraction of a tooth without any incisions through the gingiva except through the gingival sulcus. Luxation is the main surgical technique used for extracting teeth using a closed technique.
- Extraction sites are often left open to heal by second intention.
- ***Open***: creation of a mucoperiosteal flap to expose buccal surface of tooth.

Indicated for-

  - Canine teeth to ensure extraction site can be closed.
  - Multi-rooted teeth: in order to allow the teeth to be sectioned into individual rooted sections, the majority of teeth buccal bone removal facilitates a more controlled extraction. There are three roots in the maxillary fourth premolar (cat/dog), and first and second molar (dog).
  - Tooth resorption: if there are root abnormalities such as a dilaceration or apical bulb that will increase the difficulty of the extraction.

Technique

- Cats' teeth (especially with tooth resorption) fracture easily, so an open extraction technique is preferable unless there is severe periodontitis. A good luxation technique greatly facilitates feline tooth extractions.
- Always incise the gingiva first according to your planned mucoperiosteal flap design using a scalpel blade. The periosteum should then be lifted from the bone using a periosteal elevator working from crown to apex in a sweeping motion.
- Luxate the tooth: the concave surface should approximate the curve of the root; advance into sulcus and work towards to apex of the tooth on one side. Once luxation is complete along one side, move to the opposite side to luxate and this will gently push the tooth into the area of bone compression created with the first luxation procedure.
- Once there is sufficient space, an elevator can be introduced between the tooth and the bone and then gentle rotational pressure is applied in order to tear the periodontal ligament fibres and move the tooth around in the alveolus.
- Remove the tooth with fingers or forceps.

- Retained roots: radiograph to assess the amount of root left. Remove bone so that the root fragment can be visualised. Create a 'moat' around the root fragment with a small burr in order to enlarge the periodontal ligament space here. A luxator can then be reintroduced into the periodontal ligament space to extract the root using luxation.
  - Avoid pushing down on the root fragment with dental instruments as this will cause an iatrogenic intrusive luxation of the root fragment through the alveolus. These root fragments can then be very challenging to remove; clients need to be informed if removal is not possible.

NUTRITION

3.2.8 NUTRITION AND DENTAL DISEASE

- The relationship between processed (especially wet) pet foods and dental disease is the subject of much debate but few good studies.
- When choosing a diet, all the health needs of the patient should be considered, together with owner factors such as their ability of the carer to perform routine oral health care.
- Some products are marketed as main diets and others as treats aimed at reducing tartar build-up. Some are quite high in calories so main ration needs to be reduced.

SELECTED TOPIC

3.2.9 EQUIPMENT

- Good dental care does not necessarily require expensive equipment, but an appropriately sized selection of well-maintained, sharp hand tools are essential (Figure 3.2b). Dental instruments should be sharpened and sterilised between each patient.

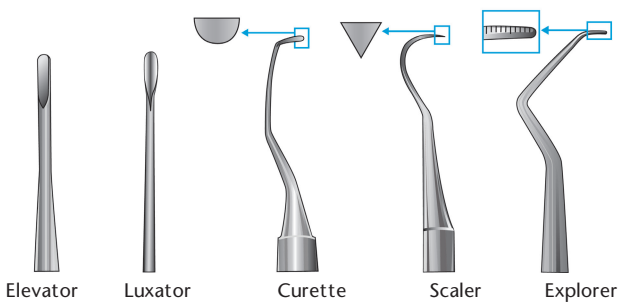


Figure 3.2b Dental instruments.

3.3 HEART AND LUNGS

3.3.1 *Prevalence and causes of cardiorespiratory disease* 3.3.2 *Checklists* 3.3.3 *Diagnostic tests and their objectives* 3.3.4 *Thoracic imaging: lungs* 3.3.5 *Thoracic radiography: heart* 3.3.6 *Drug therapy* 3.3.7 *Nutritional management in cardiac disease* 3.3.8 *Blood pressure*

INTRODUCTION

3.3.1 PREVALENCE AND CAUSES OF CARDIORESPIRATORY DISEASE

- Between 15% and 20% of sick cats and dogs presenting to general practice have cardiorespiratory disease.
- Congenital cardiac disease is relatively common, affecting between 0.5% and 1% of puppies and 0.25 and 0.5% of cats; congenital respiratory disease is rare.
- Primary bacterial causes of respiratory disease are uncommon and the presence of bacteria suggests another underlying condition that has damaged the defence system.
- For approaches to common presenting signs of cardiorespiratory disease, see 2.1 Sneezing and nasal discharge; 2.2 Coughing; 2.3 Dyspnoea. For critical care see 5.5 Dyspnoea; 5.6 Collapse.

HISTORY AND PHYSICAL EXAMINATION

3.3.2 CHECKLISTS

- On history and physical examination, there may not be a clear separation between cardiac and respiratory disease, especially in cats.
- In such cases, the history and physical examination pointers for both body systems should be used.

History: cardiac disease

- Recent changes in attitude, activity, or exercise tolerance.
- Changes in respiratory rate/effort noticed at rest.
- Encourage owner to measure sleeping respiratory rate (SRR).
- Excessive panting at exercise: is there an association with degree of exercise and weather?
- Falling over or weakness: what had the patient just done, or was doing at the time?
- Whether the patient is coughing: type, timing, progression, description.
- Check colour of mucous membranes, particularly at exercise.
- In cats, presence of a gallop, pulse rate over 200 bpm, respiratory rate over 80/min and temperature <37.5°C are strong predictors of heart disease.

History: respiratory disease

- History should be focused on differentiating between upper and lower respiratory tract disease – see 2.1, 2.2, and 2.3.

Physical examination: cardiac disease

Abnormalities in the following would tend to indicate the presence of cardiovascular disease. You should record:

- Regularity of rate and rhythm.
- Is heartbeat regularly or irregularly irregular?
- Synchrony of heartbeat with pulse.
- Pulse quality.
- Mucous membrane colour (if pale, see 2.14).

- Presence of heart murmur; focus on:
  - Systolic (common) or diastolic (rare) – all, or part of phase.
  - Point of maximum intensity.
  - Radiation.
  - Loudness (Table 3.3a).
  - Shape, quality, and pitch.

Physical examination: respiratory disease

- Observe rate, pattern, and character of breathing.
- Examine nasal area for symmetry, hair discoloration, and nasal discharge.
- Check air flow from both nares.
- Palpate the upper respiratory tract and thorax (for integrity, compressibility, and position of the apex beat).
- Gently squeeze the larynx to assess cough response.
- Auscultate the thorax and trachea for the location, intensity, and normality of breathing and cardiac sounds.
- Percuss the thorax for increased (air) and decreased (fluid, soft tissue) resonance.

DIAGNOSIS

3.3.3 DIAGNOSTIC TESTS AND THEIR OBJECTIVES

- Routine haematology and biochemistry are often of limited value.
- Infectious disease testing should be considered:
  - Cats – FeLV, FIV, feline herpes virus 1 (FHV-1), feline calicivirus (FCV).
  - Dogs – angiostrongylus, *Dirofilaria*, canine herpes virus, *Crenosoma*, parainfluenza virus.
  - On bronchoalveolar lavage (BAL) samples – mycoplasma, *Bordetella*, adenovirus.
- ***Cardiac troponin I***: looking for active myocardial damage.
- ***NT-proBNP***: looking for left ventricular stretch and overload; differentiating heart failure from respiratory disease as a cause of dyspnoea (Table 3.3b).
  - Check appropriate sample type and handling before sending.

Table 3.3a Heart murmur: grades of loudness

Grade I	Quiet: need good listening conditions to hear
Grade II	Soft but easily heard
Grade III	Moderate: similar to intensity of normal heart sounds
Grade IV	Loud: louder than intensity of normal heart sounds
Grade V	As IV but with precordial thrill
Grade VI	Very loud, precordial thrill, heard away from body wall

Table 3.3b NT-proBNP results: interpretation

Interpretation	**NT-proBNP level (pmol/l)**	
	Dog	**Cat**
Normal – heart disease unlikely	<800	<50
Elevated – heart disease possible	800–1800	50–100
Heart volume overload likely	>1800	100–270
Congestive heart failure	>2700	>270

- ***Electrocardiography***: ECG is best at assessing rhythm and type of rhythm disturbance; see 1.3.8, 1.3.9.
- ***Blood pressure***: see 3.3.8.
- ***Radiography***: best used for nasal, lung, and pleural disease; heart size; see 1.3.10.
- ***CT*** is better than radiography for nasal and lung disease.
- ***Echocardiography*** for myocardial and valvular function, congenital heart disease, pleural fluid, and mediastinal disease, B lines; see 1.3.14.
- ***Standard MRI*** is of limited value in cardiac and lower respiratory tract disease.

3.3.4 THORACIC IMAGING: LUNGS

Common lung patterns

- There are three common lung patterns. Although often mixed, there is usually one pattern that predominates and can be used to construct a differential diagnosis list.
- ***Alveolar pattern*** (Figure 3.3a) is caused by filling of the alveoli with fluid or cells, resulting in a 'fluffy' appearance that is denser towards the centre of the area, with an ill-defined edge as the affected lung merges into normal lung. In extreme cases, where all the alveoli are filled, an air bronchogram is created and the lobe becomes consolidated.
- Pulmonary oedema starts in the perihilar area (dogs) but can be anywhere in the lung fields in cats.
- ***Bronchial pattern*** (Figure 3.3b) occurs when the bronchial wall is prominent (inflammation, fibrosis, calcification) or there is peribronchial infiltration.
    - End on, bronchi appear as 'doughnuts' or 'signet rings'; longitudinal airways are described as 'tramlines'.
    - In acute bronchial disease the edge of the wall tends to be thicker, less radiodense, and less well defined.
    - In chronic disease the wall tends to be brighter and more sharply defined.

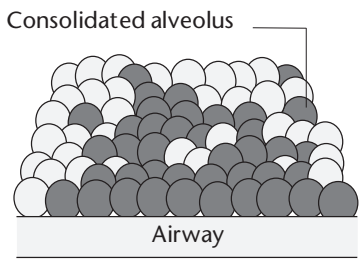


Figure 3.3a Alveolar pattern.

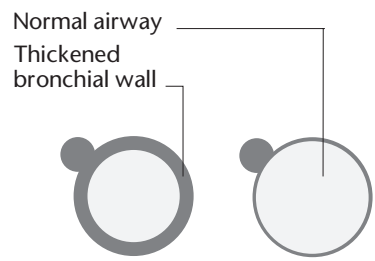


Figure 3.3b Bronchial pattern.

- ***Interstitial patterns*** are caused by diseases that affect the support tissue of the lungs. This is seen either as a diffuse 'chicken-wire' appearance, where the general lung density is increased and structures such as the blood vessels become less distinct, or as a nodular pattern such as occurs with metastatic disease. Interstitial density tends to increase with age.

Radiographic signs of pleural disease

- Free pleural fluid usually causes separation of the lung from the body wall by a soft-tissue density. Lung scalloping (lateral view) or rounding (dorsoventral view) tends to be associated with more chronic disease.
- Pneumothorax causes the lungs to collapse centrally, so dense soft tissue is surrounded by black air that, even with windowing, has no lung structures within. In deep chested dogs, peripheral lung fields can appear black, but a faint lung pattern is present.
- Finding abdominal contents in the pleural space indicates ruptured diaphragm.

Lung ultrasound

- Ultrasound of the lungs to detect fluid/cellular material present in the alveoli (wet lungs) is becoming more prevalent. In dyspnoeic patients it is less stressful than radiography or CT.
- In a normal patient 'A lines' are seen as the lung surface moves horizontally across the screen. With wet lungs 'B lines' (lung rockets, comet tails) (Figure 3.3c) are seen as reverberating lines away travelling into the lung depth.

3.3.5 THORACIC RADIOGRAPHY: HEART

Common heart changes.

- Heart size can be assessed in a variety of ways:
  - 2.5–3.5 rib spaces and two-thirds thoracic height on lateral.
  - Two-thirds thoracic width at the fifth intercostal space on the dorsoventral (DV) view.
  - Vertebral heart score. Long- and short-axis heart dimensions are transposed onto the vertebral column and recorded as the number of vertebrae, beginning with the cranial edge of T4; the vertebral heart score is the sum of these values (Figure 3.3d) and is normally 8.5–10.5 (dog); 6.7–8.5 (cat).
  - These criteria are not perfect: use judgement.

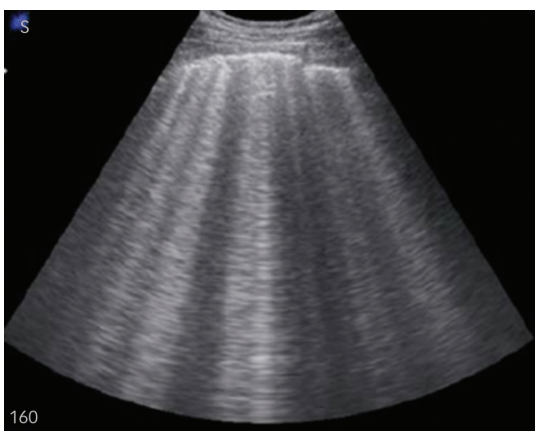


Figure 3.3c B-lines on thoracic ultrasound associated with pulmonary oedema.

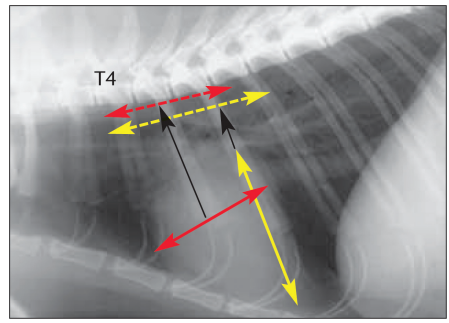


Figure 3.3d **Vertebral heart score.** VHS = sum of the heart's short axis dimension (red arrows) and long axis dimension (yellow arrows) calculated as the number of vertebrae starting at T4. In this cat VHS = 7.5.

- Note that heart shape varies with breed and a quick glance can be misleading, e.g. the heart always looks big in small terrier breeds; Labradors often have a rounded heart shape.
- Small heart: consider dehydration, over-inflation of lungs, shock, pneumothorax, obstruction to venous flow.
- Normal heart: consider atrial or small ventricular septal defect, mild valvular or myocardial disease, early bacterial endocarditis, constrictive pericarditis, neoplasia, congestive heart failure on diuretics.
- Large heart: consider volume overload associated with severe valvular disease or myocardial failure, marked HCM, pericardial effusion.
- Increased chamber size.
    - Left atrial enlargement is usually seen as a straight caudal border to the heart (dog) and increased density at the carina due to volume overload and is often associated with pulmonary oedema.
    - Right side enlargement causes increased sternal contact, secondary to tricuspid/pulmonic valvular disease, pulmonary hypertension.

Pulmonary vasculature

- Cardiovascular disease may lead to changes in pulmonary vasculature. Criteria for normality are:
    - In a normal individual arteries and veins are of a similar diameter.
    - Lateral view: arteries lie dorsally, and veins lie ventral to the bronchus. Width of cranial pulmonary vessels normally less than width of the proximal third of rib 4.
    - Dorsoventral view: the arteries lie lateral to the vein. Normally less than the diameter of rib 9 as the vessel crosses the ribs.

TREATMENT

3.3.6 DRUG THERAPY

- ***Cardiac disease***: see Table 3.3d. Therapy for cardiac disease should be directed as far as possible at the cause of reduced cardiac output.
    - Many drugs can be hypotensive, so take care when starting multiple therapies at the same time.
    - Pimobendan has been shown to extend disease free period and survival time in dogs with stage B2 myxomatous mitral valve disease and Dobermans with occult cardiomyopathy.
- ***Coughing and dyspnoea***: see Table 3.3e. Successful treatment is very dependent on an appropriate diagnosis. Therapy based on clinical signs will often fail to produce sustained improvement in chronic disease.

Table 3.3c Nutritional management in cardiac disease

Asymptomatic disease	Mild–moderate disease	Severe disease
Mild sodium restriction	Moderate sodium restriction	Increased sodium restriction
Maintain optimal BCS	Maintain optimal BCS	Enhance palatability to maintain BCS
n-3 PUFA	n-3 PUFA Maintain serum magnesium and potassium levels in reference range	n-3 PUFA Benefits of taurine, carnitine, co-enzyme Q, B-vitamins and L-arginine supplementation, where specific deficiency has not been documented, are unknown

BCS: body condition score; PUFA: polyunsaturated fatty acid

NUTRITION

3.3.7 NUTRITIONAL MANAGEMENT IN CARDIAC DISEASE (TABLE 3.3C)

- Concerns have been raised about dogs developing dilated cardiomyopathy (DCM) when fed a grain free, legume-rich diets.
- Taurine deficient diets are known to cause DCM in cats – vegetarian and vegan diets for cats MUST be taurine supplemented.
- See Table 3.3c for general recommendations. The true value of dietary intervention has been difficult to document.

SELECTED TOPIC

3.3.8 BLOOD PRESSURE

- Both hypertension and hypotension can cause life-threatening disease.
- ***Measurement***: normal systolic BP (SBP) measured by Doppler is 120–150 mmHg. Oscillometric methods are less reliable in conscious patients, and tend to underestimate BP.
- Note that BP measurements in conscious animals are inherently unreliable: situational hypertension ('white coat effect') can be as much as 50 mmHg, especially in cats.
- Substantial inter-breed variation in normal BP in dogs.
- In anaesthetised/recumbent animals, BP trends are more important than absolute values (unless extreme).
- Diagnosis of hypertension should therefore only be made after multiple measurements, on more than one occasion, showing high BP. Hospitalisation and repeated measurements in a day can habituate some patients to give more reliable readings.
- If hypertension is noted, the patient should also be examined for evidence of clinical effects or target organ damage (TOD) such as:
  - Retinopathy or intraocular haemorrhage.
  - Chronic or acute renal failure.
  - Signs of hyperadrenocorticism, diabetes mellitus, or hyperthyroidism.
  - Unexplained neurologic signs.
  - Left ventricular hypertrophy.
- ***Classification***
  - Normotensive (minimal TOD risk) SBP <140 mmHg
  - Prehypertensive (low TOD risk) SBP 140–159 mmHg
  - Hypertensive (moderate TOD risk) SBP 160–179 mmHg
  - Severely hypertensive (high TOD risk) SBP ≥180 mmHg
- ***Approach*** to hypertension: (Figure 3.3e). This is based on systolic BP measured by the Doppler method. Ideally, diagnosis is based on multiple measurements obtained at more than one visit but if BP is very high especially if there is TOD then start treatment after single measurement.

- ***Management***: hypertension can be managed by oral therapy as follows:
  - Initial treatment:
    - Amlodipine 0.625–1.25 mg/cat q24 hr–q12 hr.
    - Telmisartan 2–3 mg/kg PO q24 hr.
    - Combination if no response.
  - Acute life threatening – nitroprusside continuous rate infusion (CRI); hydralazine PO.

Table 3.3d Commonly-used drug therapy for cardiac disease

Drug	Dose rate (dog)	Dose rate (cat)	Comment
Furosemide*§ (diuretic) 50 mg/ml inj; 20/40 mg T; 4–10 mg/ml S (NL)	Acute pulmonary oedema 1–2 mg/kg q2–6 hrs IV or IM until stable. Can be given SC. Oral maintenance therapy 1–4 mg/kg q12–24hr	Acute pulmonary oedema 1–2 mg/kg q4–8 hrs IV or IM until stable. Can be given SC. Oral maintenance therapy 0.5–2 mg/kg q8–24hr	Use lowest dose possible. If high doses (>2–4 mg/kg/day) consider additional diuretic. Continuous rate infusion (0.1–0.5 mg/kg/hr) in acute cases may be better than pulse-dosing
Torasemide (diuretic) 0.75, 3, and 7.5mg tablets	0.1–0.6 mg/kg PO q24 hr	0.1-–0.6 mg/kg PO q24 hr 0.05–0.3 mg/kg PO q12 hr	Often used as rescue when furosemide therapy failing. Use 1/20th dose of torasemide, i.e. if on 10 mg/kg/day furosemide start at 0.5 mg/kg/day torasemide. Similar risks to furosemide – hypokalaemia, hyponatraemia, dehydration, AKI, ototoxicity
Spironolactone* (diuretic) 10, 40, 80 mg T; 20 mg/ml S (NL)	2–4 mg/kg orally once daily	2–4 mg/kg orally once daily	Potassium-sparing diuretic, synergistic with furosemide; rarely sufficient alone
ACE inhibitors Benazepril* 5, 20 mg T	0.25–0.5 mg/kg q24hr	0.25–0.5 mg/kg q24hr	All have similar action and act as a balanced vasodilator, reduce renin–angiotensin–aldosterone system activation and improve vascular remodelling. Liver/kidney excretion varies between agents. Tend to reduce blood pressure: use with care in hypovolaemic or hypotensive cases
Enalapril* 1, 2.5, 5,10, 20 mg T	0.25–1 mg/kg q12–24hr	0.25–1 mg/kg q12–24hr	
Imidapril* 75, 150, 300 mg powder	0.25 mg/kg q24hr	0.25 mg/kg q24hr	
Ramipril* 1.25, 2.5, 5 mg T	0.125 mg/kg q24hr	0.125 mg/kg q24hr	
Pimobendan* (inodilator) 1.25, 2.5, 5 mg T	0.1–0.3 mg/kg q12hr one hour before food	1.25 mg/cat q12hr one hour before food	Positive inotrope sensitizing the myocardium to calcium, without significant increase in oxygen demand. Vasodilation mediated by phosphodiesterase III activity

Drug	Dose rate (dog)	Dose rate (cat)	Comment
Diltiazem§ (calcium channel blocker) 10 mg T (60 mg T [NL]) Licensed for treatment of hypertrophic cardiomyopathy in cats Used to manage atrial fibrillation and other supraventricular tachycardias in dogs	0.5–2.0 mg/kg PO q8hr	0.5–2.5 mg/kg PO q8hr	Slow-release tablets have been used, but pharmokinetics are not defined and may have hepatotoxicity in cats. Acts to interfere with calcium movement and has a negative inotropic activity; retards atrio-ventricular conduction and dilation of the large arteries
Digoxin 0.0625, 0.125, 0.25 mg T; 50 µg/ml S	0.22 mg/m^2 PO q12hr	10 µg/kg PO q24–48hr [rarely used in cats]	Unlikely to have significant positive inotropic activity. Most useful in slowing heart rate in atrial fibrillation. Narrow therapeutic index; serum monitoring advised after 5–7 days use
Beta adrenergic blockers Atenolol 25, 50, 100 mg T; 5 mg/ml S	0.5–2 mg/kg PO q12hr	6.25–12.5 mg/cat PO q24hr	Negative chronotropic and inotropic activity in cardiac arrhythmia (both supra ventricular and ventricular). Use in congestive heart failure controversial; requires very careful patient selection and intro duction
Propranolol 1 mg/ml inj; 10, 40, 80, 160 mg T	0.02–0.08 mg/kg slow IV over 5 min q8hr, 0.25–1.5 mg/kg PO q8hr	0.04–0.06 mg/kg slow IV over 5 min q8hr, 2.5–5 mg/kg PO q8hr	
(Sotalol, metoprolol, carvedilol) Treatment of tachyarrhythmias, hypertrophic obstructive cardiomyopathy (cats), systemic hypertension, aortic or pulmonic stenosis, chronic heart failure	See formulary for dose rates	See formulary for dose rates	See formulary for contraindications and side-effects

T = tablet; S = syrup; inj = injectable

* Licensed for use in dogs; § licensed for use in cats (UK); other drugs are used off-licence; NL = non-licensed

- Failure to respond:
  - ACE inhibitors.
  - Atenolol 0.5–2 mg/kg q12 hr.
  - Hydralazine 0.5 mg/kg (dog) 2.5 mg (per cat) and increase.

Table 3.3e Commonly-used drug therapy for coughing and dyspnoea

Drug	Dose rate (dog)	Dose rate (cat)	Comment
Mucolytic Bromhexine*§ (1% powder)	2–2.5 mg/kg PO q12hr	1 mg/kg PO q24hr	Bronchial secretolytic making expectoration easier; efficacy difficult to assess
Cough suppressant opiates Morphine (wide variety of formulations and strengths)	Injectable: 0.5 mg/kg SC q6–8hr	Injectable: 0.5 mg/kg SC q6–8h	Can cause sedation, respiratory depression and constipation; contraindicated if coughing productive
Codeine 3 mg/ml S; 15, 30, 60 mg T	1–2 mg/kg PO q6–12hr	0.3–0.5 mg/kg PO q8–12hr	
Butorphanol* 10 mg/ml inj; 5, 10 mg T	0.05–0.1 mg/kg s/c, IM, IV q6–12hr; 0.5–1 mg/kg PO	0.05–0.1 mg/kg SC, IM, IV q6–12hr; 0.5–1 mg/kg PO q6–8hr	
Diphenoxylate 2.5 mg tablets with 0.025 mg atropine	0.2–0.4 mg/kg PO q8–12 hr	0.05–0.1 mg/kg PO q12 hr with care	Antidiarrhoeal medication with good antitussive activity. Can cause sedation, constipation and ileus. Atropine present to discourage abuse
Bronchodilators Etamiphylline*§ 140 mg/ml inj; 100, 200, 300 mg T	15 mg/kg SC, IM q8hr; 10–20 mg/kg PO q8hr	15–25 mg/kg SC, IM q8hr; 100 mg/cat PO q8hr	CNS and cardiovascular stimulation, mild diuretic action
Theophylline* 100, 200, 500 mg SR capsule	15–20 mg/kg PO q12–24hr	20–25 mg/kg PO q24hr	
Terbutaline 0.3 mg/ml S; 5 mg T	1.25–5 mg/dog PO q8–12hr	0.3–1.25 mg/kg PO q8–12hr	
Antihistamines Many types, e.g. chlorphenamine 10 mg/ml inj; 4 mg T; 0.4 mg/ml S	4–8 mg/dog PO q8–12hr	2–4 mg/cat PO q8–12hr	Side-effects depend on drug used; can cause sedation and other anticholinergic activity; efficacy in allergic respiratory disease often poor. Second-generation antihistamines have fewer side-effects but have been less used in dogs and cats
Clemastine 1 mg T	0.1 mg/kg PO q12hr	0.05–0.1 mg/kg PO q12hr	
Anti-inflammatories Prednisolone	0.5 mg/kg PO – anti-inflammatory 1–2 mg/kg PO – immunosuppressive	Prednisolone (or other cortico steroids) useful in allergic and inflammatory airway disease, but can cause muscle wasting and tachypnoea; *contraindicated* in bacterial pneumonia and viral respiratory disease	

Drug	Dose rate (dog)	Dose rate (cat)	Comment
NSAIDs	see 4.2.4 for options and dose rates	NSAIDs useful in some nasal disease, viral upper respiratory tract disease; *contraindicated* in bronchoconstriction	
Antimicrobials	see 3.13.4 for appropriate antimicrobial choice	*Bordetella*, *Pseudomonas* and *Streptococcus zooepidemicus* are the primary bacterial invaders of the respiratory tract	
Inhaled therapy Other corticosteroid-based inhalers available Fluticasone propionate (Flixotide)	50–250 µg q12–24hr	50–250 µg q12–24hr	Spacing device required – if multiple actuations required, should be given as one dose multiple times. More expensive than oral therapy, but fewer side-effects in long-term use. Salbutamol can cause tachycardia, tremor and CNS stimulation
Salbutamol (Ventolin)	100 µg q4–6hr	100 µg q4–6hr	

T = tablet; S = syrup; SR = slow release; inj = injectable; SC = subcutaneous, IM = intramuscular; IV = intravenous

* Licensed for use in dogs; § licensed for use in cats

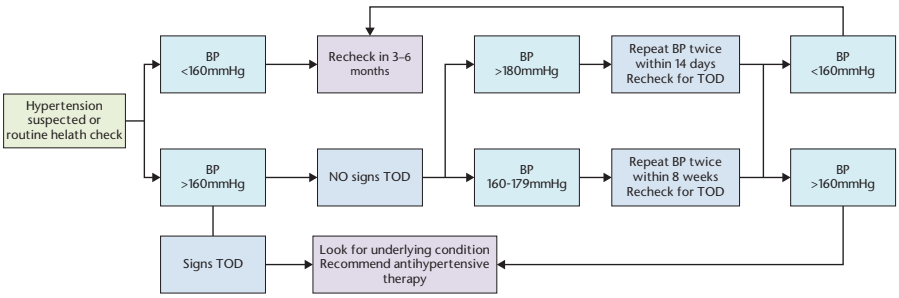


Figure 3.3e Algorithm outlining the approach to managing systolic blood pressure in cats and dogs. From Agiemo et al. ACVIM Consensus Statement 2018.

3.4 DIGESTIVE TRACT

3.4.1 *Acute/chronic gastrointestinal disease* 3.4.2 *Key questions and examination tips* 3.4.3 *Preliminary diagnostic tests* 3.4.4 *Abdominal imaging* 3.4.5 *Drug treatment* 3.4.6 *Dietary approach to intestinal disease* 3.4.7 *Pancreatitis*

INTRODUCTION

3.4.1 ACUTE/CHRONIC GASTROINTESTINAL DISEASE

- Gastrointestinal (GIT) disease presents in both acute and chronic forms; for approaches see 2.4, 2.5, 2.6, and 2.12.
- With ***acute disease***, the key question is whether the problem is self-limiting. In self-limiting cases there will be a short history and often known exciting cause; the patient

will be systemically well, often with good appetite, and there will be negative physical findings. Where the problem is severe, the patient is usually unwell with positive physical findings indicative of GIT disease: such cases require immediate investigation.

- For ***chronic or recurrent disease***, the key is to follow a logical path of investigation and treatment (preferably monotherapy) so that effective management is eventually achieved.

HISTORY AND PHYSICAL EXAMINATION

3.4.2 KEY QUESTIONS AND EXAMINATION TIPS

- ***History*** is crucial, especially in chronic disease where there may be minimal physical findings. Description of the signs when the disease first began is often the most helpful.
  - In patients that are 'vomiting', it is essential to be certain the owner is not describing regurgitation, gagging, dysphagia, expectoration, or retching. On rare occasions, patients will show a combination of signs.
  - Regurgitated material (alkaline) can be distinguished from vomitus (acidic) by measuring the pH, using litmus paper.
  - In vomiting cases, the relationship to feeding and the nature of the vomitus are important.
- ***Physical examination***: improved access to the abdomen can sometimes be achieved by raising the front legs.
  - Limited information can be gained from large breed and overweight/obese dogs.

! The intestine of thin animals can often feel abnormally prominent, but in many cases, this is a normal finding due to the lack of abdominal fat.

- ***Faecal score chart*** (Figure 3.4a): can be useful for in-hospital description by care staff, and for home use by owner.
- ***Small- vs large-bowel diarrhoea***: small-intestinal dysbiosis can cause large-intestinal signs. Localisation of disease according to faecal characteristics is less reliable in cats.
  - Typically, small intestinal diarrhoea is characterised by increased fatty stools, melaena, and undigested food. Large bowel diarrhoea is characterised by increased frequency, urgency, tenesmus, mucus, and fresh blood.

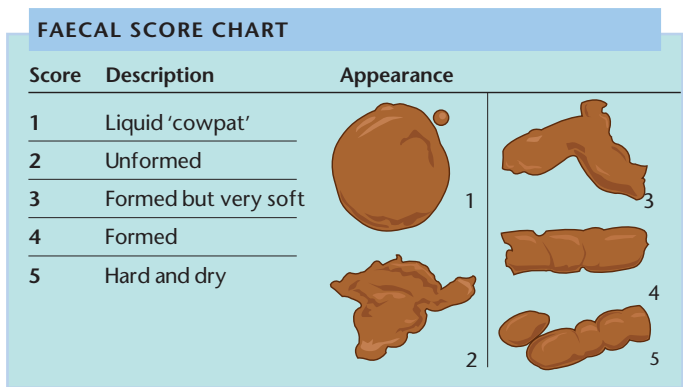


Figure 3.4a Faecal score chart.

DIAGNOSIS

3.4.3 PRELIMINARY DIAGNOSTIC TESTS

- Full faecal examination is essential in cases of chronic diarrhoea – parasitology, culture for pathogenic bacteria, viral testing as appropriate.
- Routine haematology and biochemistry are often of limited value in chronic disease, but important in acute disease where the patient is systemically unwell. In such cases it will help to exclude non-GIT disease as the underlying cause, as well as documenting any metabolic consequences of the disease process. Chronic disease can lead to anaemia associated with haemorrhage, hypoproteinaemia, and hypocholesterolaemia.
- GIT-focused blood tests include:
  - TLI, PLI, amylase, and lipase – see pancreatitis (3.4.7).
  - Folate and cobalamin – changes outside of the reference range can be supportive of GIT disease.
  - Hypocobalaminaemia requires supplementation.
  - A basal cortisol should be considered in all dogs with chronic disease. Values <55 nmol/l may indicate hypoadrenocorticism.
  - A thyroid hormone level test is appropriate in older cats.

3.4.4 ABDOMINAL IMAGING

Radiographic changes in common GIT diseases

For basic abdominal radiographic anatomy see 1.3.12.

- Megaoesophagus
  - Wall is highlighted by intraluminal gas (Figure 3.4b).
  - Heart/trachea is displaced ventrally.
  - Check for aspiration pneumonia (dependent parts of lobes, especially right middle and left cranial) and mediastinal mass.
- Gastric dilation/torsion
  - Torsion shows as a gas-filled stomach usually with linear soft-tissue density crossing the stomach on a lateral view.
  - Gas may be present in intestines. The pylorus is displaced dorsal, cranial, and to left (Figure 3.4c).
- Small intestinal obstruction
  - Small intestinal loops are dilated proximal to obstruction (greater than diameter of large bowel).
  - Colon may be empty.
  - Gravel signs may be present.

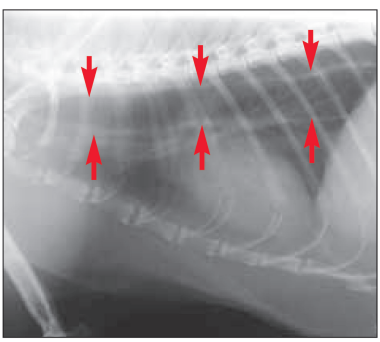


Figure 3.4b Megaoesophagus.

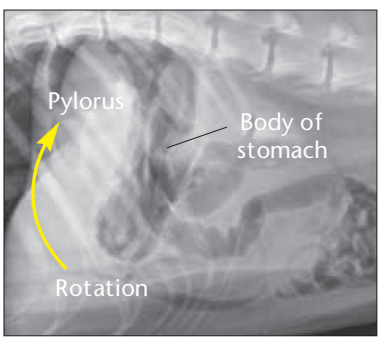


Figure 3.4c Gastric dilation-volvulus.

- Peritonitis
  - Septic vs non-septic:
    - Check with ultrasound; septic fluid tends to be granular.
    - Microscopic examination of fluid: bacteria = septic.
  - Regionalised vs generalised.
  - Loss of sharp outline of abdominal organs.
  - Serosal surfaces not clearly seen.
- ***Ultrasound***: complete ultrasound of the GIT is challenging and requires an experienced ultrasonographer. However, useful information can still be gained looking for evidence of free fluid, dilation of the stomach or bowel, and gross changes in wall thickness or layering.

TREATMENT

3.4.5 DRUG TREATMENT

- Drugs commonly used in the management of GI disease are listed in the Table 3.4a.

NUTRITION

3.4.6 DIETARY APPROACH TO INTESTINAL DISEASE

- ***Adverse reactions to food*** can be immunologically based or non-immunologic (pharmacologically active micronutrients such as caffeine or theophylline, histamine in cheap fish as well as preservatives, additives and dyes and toxic contamination from bacteria or chemicals).
- ***Dietary hypersensitivity***: typical signs are weight loss, vomiting, and diarrhoea; 10% of cases have pruritic skin disease. Investigate by elimination diet as follows.
  - Diet should contain a novel protein and carbohydrate source. It can be commercial (single source protein or hydrolysed [more effective]) or home-cooked (e.g. boiled white fish and sweet potato).
  - Strict adherence is essential. Most cases likely to respond will start to do so in 1–2 weeks.
  - Responders should ideally be confirmed by rechallenge.
- ***Acute vomiting and diarrhoea***: GI rest not shown to be of value in patients that are systemically well, consider a palatable, low fat, highly digestible, low residue diet. For home-cooked food, protein:carbohydrate ratio ≈ 1:1 (cat); 1:2 (dog). Malnutrition can occur with frequent starvation; feeding through episodes is advised.
- **Protein losing enteropathy (PLE)**: give increased levels of high biological value protein, increased carbohydrates as an energy source and reduced fat (<4% DM). Give increased fat-soluble (A, D, and E) vitamins.
- ***Colitis***: similar diets as for dietary hypersensitivity; for chronic cases, increasing fibre may help.
- ***Faecal incontinence***: manage with low residue diets.
- ***Constipation*** cannot be treated by diet. However, the likelihood of recurrence can be reduced by using insoluble fibre to increase faecal bulk and stimulate defecation, while producing a softer stool.
  - 4 tsp of a high-fibre breakfast cereal per 100 g of food increases the fibre content of a diet by ≈10%.
- Probiotics (cultured bacteria) and prebiotics (soluble fibre, e.g. fructo-oligosaccharide) have a role in the management of chronic GIT disease. Their precise mechanism(s) of action are unclear. Avoid natural yoghurts, because of the level of lactose.

Table 3.4a Drugs for management of GI disease

Drug	Dose (mg/kg)	Side-effects
H_2 blockers *Cimetidine (T, S, inj, SC, IM, IV)	5–10 q8hr (dog PO,inj) 2.5–5 q12hr (cat PO, inj)	Rare: hepatic, renal and thrombocytopenia
Ranitidine (T, S, inj, SC, IM, IV)	2 q8–12hr (dog PO, inj) 2.5 (inj) 3.5 (PO) (cat)	
Famotidine (T)	0.5–1 q12–24hr (PO)	
Proton pump inhibitors Omeprazole (T, capsule, inj, IV)	0.5–1.5 q12hr (dog PO), 0.75–1 q12hr (cat PO) 1 q24hr (dog/cat inj)	Nausea; diarrhoea. Use for no more than 8 weeks
Pantoprazole (I, IV)	07–1 IV over 15 min	As omeprazole
Protectants Sucralfate (T, L)	25–50 q6–8hr (dog PO) 250/cat q8-12hr (PO)	Constipation
†Kaolin-based pastes	See product	
Antiemetics *Maropitant (T, inj, SC)	2 q24hr (dog PO) 1 q24hr (dog inj)	Injection can be painful
†Metoclopramide (T, L, inj, SC, IM, IV)	0.2–0.5 q6–8hr (PO) 0.2–0.5 q6–8hr (inj)	Neurologic: can be severe
Ondansetron (T, I, rectal IV)	0.5 loading + 0.5/hr CRI 0.5–1 q12–24hr PO	Rare – constipation headaches (man)
Prokinetics Ranitidine (T, S, inj, SC, IM, IV)	2 q8–12 (dog PO, I) 2.5 q12 (I); 3.5q12 (cat PO)	As cimetidine
†Metoclopramide (T, L, inj, SC, IM, IV)	As above, also 1–2 q24hr CRI	As above
Erythromycin (T, L)	0.5–1 q8hr	Rare at this dose
Motility modifiers Diphenoxylate (T)	0.05–0.1 q6–8hr (dog po)	Sedation, constipation, ileus
Loperamide	0.04–0.2 q8–12hr (po)	Constipation; Toxicity in collie types

* Injectable licensed for dogs and cat; tablets dogs only; † considered a nutraceutical
T = tablet; S = syrup; L = liquid; inj = injectable; SC = subcutaneous; IM = intramuscular; IV = intravenous

SELECTED TOPIC

3.4.7 PANCREATITIS

- Pancreatitis can occur as a primary problem, or secondary to another disease.
- Sick hospitalised patients are at risk of developing pancreatitis, even if it is not present on admission.
- Mortality in acute pancreatitis cases is likely to be 25–50%.
- ***Presenting signs*** might include:
  - Acute, dogs: anorexia, vomiting, diarrhoea, abdominal pain, fever.
  - Acute, cats: lethargy, anorexia, jaundice, fever, and less commonly vomiting and diarrhoea.
  - Chronic, dogs: anorexia, vomiting, weight loss, jaundice.

- Chronic, cats: anorexia, weight loss, vomiting, jaundice.
  - Chronic pancreatitis in cats may eventually lead to diabetes mellitus and/or exocrine pancreatic insufficiency.

! *Absence of abdominal pain does not reliably rule out pancreatitis.*

- Laboratory changes
  - TLI, amylase and lipase are insensitive.
  - PLI and DGGR (1,2-o-dilauryl-rac-glycero-3-glutaric acid- (6′-methylresorufin) ester) lipase are relatively much more sensitive and specific but note that false positive and false negative results do occur in both acute and chronic pancreatitis.
- Acute cases need full haematology and biochemistry.

IMAGING

- ***Acute***: radiographic changes are those associated with focal or generalised peritonitis and/or cranial abdominal mass. Ultrasound is more specific and aids aspiration of fluid from the pancreas and small pockets of fluid around it, as well as assessing the level of biliary obstruction and other disease.
- ***Chronic***: requires ultrasound by an experienced ultrasonographer, most common changes are increased thickness and changes in parenchymal echogenicity. Sensitivity and specificity are quiet low.

TREATMENT

- Starvation of acute pancreatitis cases is no longer recommended.
- ***Acute***: treatment should be individualised according to patient's problem list, but may include the following:
  - Pain relief (opioids not NSAIDs) and fluid therapy (Hartmann's).
  - Plasma and/or albumin.
  - Gastric protection, antiemetics and prokinetics – omeprazole, ondansetron, CRI metoclopramide.
  - Antimicrobial (IV – metronidazole, amoxicillin clavulanate).
  - Vitamins B12 and K.
  - Encouragement to eat low-fat food, e.g. baby rice and Bovril or low fat, highly digestible food.
    - Tube placement improves outcomes; if vomiting, will need to be a jejunostomy tube.
  - Regular monitoring is required of proteins, haematocrit, electrolytes, liver.
- ***Chronic***: low-fat (but not high fibre) diets are beneficial in dogs but have not been demonstrated to be useful in cats.

3.5 LIVER AND BILIARY TRACT

3.5.1 *Presentation* 3.5.2 *Principal functions of the liver* 3.5.3 *Major signs of liver dysfunction* 3.5.4 *Signs on physical examination* 3.5.5 *Interpretation of blood tests* 3.5.6 *Approach to raised liver enzymes* 3.5.7 *Further diagnostics* 3.5.8 *Choice of therapies* 3.5.9 *Dietary approach to reduced liver function* 3.5.10 *Portosystemic shunts* 3.5.11 *Gall bladder disease* 3.5.12 *Liver neoplasia*

INTRODUCTION

3.5.1 PRESENTATION

- Raised liver enzymes are commonly found on routine blood screening. Appropriate investigation and management is important, as many patients do not have clear signs associated with hepatobiliary pathology.
- Jaundice (see 2.16) is a worrying presenting complaint; accurate diagnosis is essential to allow prompt treatment.

3.5.2 PRINCIPAL FUNCTIONS OF THE LIVER

- Understanding the anatomy of the liver (Figure 3.5a) is important, particularly when interpreting histology reports.
- Besides metabolism of food, hormones, drugs, and toxins, the liver also has haemopoietic and immune functions, as well as producing enzymes and clotting factors and storing iron, vitamins, glycogen, and so on.

HISTORY AND PHYSICAL EXAMINATION

3.5.3 MAJOR SIGNS OF LIVER DYSFUNCTION

- Jaundice (2.16), hypoglycaemia, hypoalbuminaemia, ascites, hepatic encephalopathy (episodes of stupor/disorientation with or without apparent blindness, especially postprandially), bleeding (5.2), PU/PD (2.8), lethargy, inappetence (2.4), weight loss/poor growth (2.12).

3.5.4 SIGNS ON PHYSICAL EXAMINATION

- Unless the liver is significantly enlarged, it is rarely palpable.
- Gross ascites can be diagnosed on ballottement; mild to moderate ascites and focal fluid may not be easily discernible on physical examination.
- Biliary obstruction usually associated with jaundice (bilirubin >50 µmol/l). If secondary to pancreatic disease may cause cranial right quadrant pain.

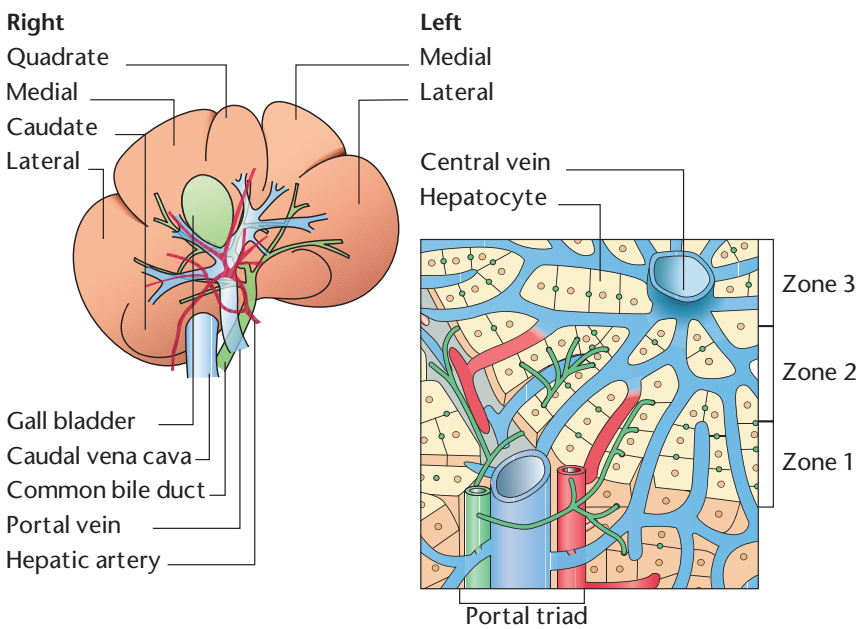


Figure 3.5a Liver and biliary anatomy.

DIAGNOSIS

3.5.5 INTERPRETATION OF BLOOD TESTS

Biochemistry is an important initial investigation for liver disease. Tests that indicate liver damage (ALT, ALKP) should be distinguished from tests of liver function (bile acids, ammonia).

- See Table 3.5 and 1.3.1.

3.5.6 APPROACH TO RAISED LIVER ENZYMES

- For an approach to raised ALKP and ALT in dogs, see Figure 3.5b.
- Mild to moderate increases in liver enzymes are of greater clinical significance in cats and generally warrant further investigation. This is because in cats:
  - Cellular levels of liver enzymes are lower.
  - Half-life is shorter.
  - There is no steroid-induced isoenzyme of ALKP.

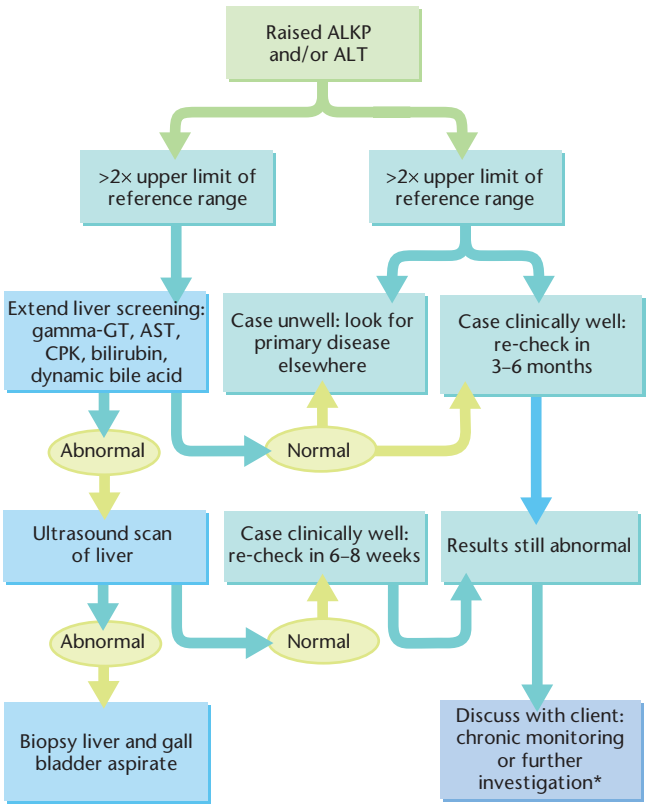
Table 3.5 Interpretation of blood tests

Parameter	Description	Non-hepatic causes of change
ALKP increase	Inducible group of isoenzymes primarily associated with biliary disease. $T_{1/2}$ 70 hrs (dog) 6 hrs (cat). Smaller increase seen in cats with hepatobiliary disease	Growing animals, enteritis, pancreatitis, bone disease, endocrine disease, drugs (esp. corticosteroids [dog])
ALT increase	Cytosolic enzyme released on damage to hepatocytes $T_{1/2}$ 60 hrs (dog) <24 hrs (cat). Will show chronic increases as liver regenerates	Endocrine disease, hypoxia, enteritis, pancreatitis, peritonitis, toxins, trauma, drugs
AST increase	Cytosolic enzyme released on damage to hepatocytes $T_{1/2}$ 5 hrs (dog) 80 min (cat)	Muscle damage/ inflammation, red cell damage
γ-GT increase	Bile ducts and perilobular tissue (dog). Increase due to *de novo* synthesis, regurgitation and from cell membrane. Increase more in biliary vs. parenchymal disease	Corticosteroids (dog) and anticonvulsants, pancreatitis, enteritis
GLDH	Mitochondrial enzyme increases in hepatic necrosis (dog)	
Bilirubin increase	Bilirubin can be elevated due to excess production (pre-hepatic), an inability of the liver to process bile (hepatic) or obstruction to bile flow (post-hepatic)	
Bile acids increase	Synthesized by hepatocytes and secreted in bile. 95% enterohepatic circulation and should fall to baseline within 2 hours	Jaundice, elevated glucocorticoids, pancreatic disease
Albumin	Severe impairment of functional liver mass	Urinary, intestinal or 3rd space loss
Ammonia increase; urea decrease	Produced from nitrogen metabolism and intestinal bacteria; conjugated in liver to form urea. Increases indicate severe hepatic impairment. Volatile, so rapid measurement essential; false positives if ammoniacal cleaners recently used	
Cholesterol change	Tends to be increased with severe intrahepatic cholestasis, and decreased in chronic severe hepatocellular disease	

ALKP = Alkaline phosphatase; ALT = alanine aminotransferase; AST = aspartate aminotransferase; γ-GT = gamma glutamyltransferase; GLDH = glutamyl dehydrogenase

3.5.7 FURTHER DIAGNOSTICS

- ***Radiography*** is generally limited to assessing liver size.
- For normal liver size:
  - The gastric angle is between vertical and parallel to ribs.
  - The left lateral lobe of the liver has a sharp angle and is just within/beyond the costal arch.
- Lobar masses may be visible as they will displace other abdominal organs caudally.
- ***Ultrasound*** is usually more sensitive (though not more specific) than radiography and allows assessment of the biliary tract. Diffuse disease can be difficult to assess. In older dogs, benign nodular hyperplasia can mimic neoplasia.
- ***Aspiration/biopsy*** – clotting times should be checked before biopsy. Biopsies obtained at laparoscopy/laparotomy preferred in diffuse disease; ultrasound-guided more valuable if focal or deep lesion present. Aspirates are useful in diffuse disease such as hepatic lipidosis, lymphoma and hepatitis. Take bile aspiration for culture if bacterial cholangitis suspected.


Raised ALKP and/or ALT
>2× upper limit of reference range
>2× upper limit of reference range
Extend liver screening: gamma-GT, AST, CPK, bilirubin, dynamic bile acid
Case unwell: look for primary disease elsewhere
Case clinically well: re-check in 3–6 months
Abnormal
Normal
Ultrasound scan of liver
Case clinically well: re-check in 6–8 weeks
Results still abnormal
Abnormal
Normal
Biopsy liver and gall bladder aspirate
Discuss with client: chronic monitoring or further investigation*

* Further investigation is indicated in breeds predisposed to chronic hepatopathy, e.g. Doberman. Monitoring is advised in breeds known to have chronically high enzymes without clear pathology, e.g. Scottish terrier.

Figure 3.5b Algorithm illustrating an approach to responding to raised liver parameters in dogs.

TREATMENT

3.5.8 CHOICE OF THERAPIES

- Acute liver disease is managed symptomatically and supportively. Coagulopathies and embolism are more likely than in chronic disease.
- Chronic liver disease in dogs and cats has relatively few specific treatments. Doses of potentially toxic drugs should be reduced as much as possible, and alternative therapies sought.
- ***Antimicrobials*** are indicated if proven infection or in severe dysfunction/portosystemic shunts: give ampicillin or amoxicillin 10–20 mg/kg PO q8–12 hr.
- ***Vitamin K_1*** should be given to patients at risk of bleeding: 0.5–1 mg/kg SC or PO initially q12 hr.
- ***Diet and nutraceuticals***
  - Diet is indicated if there is hepatic dysfunction (3.5.9).
  - S-adenosyl methionine replenishes antioxidant capacity and aids metabolite detoxification. Clinical benefit in chronic disease has been difficult to prove. 20 mg/kg PO q24 hr. Must be enteric-coated, bioequivalence varies between products.
  - Vitamin E (D-α-tocopherol) is an antioxidant; give 4 mg (6iU)/kg PO q24 hr.
  - N-acetyl cysteine protects against oxidant injury in acute injury. Give 140 mg/kg IV in 5% dextrose over 15–20 minutes.
  - Silybin phosphatidylcholine (silymarin; milk thistle) is an antioxidant, benefit unproven; dose rate 5–10 mg/k PO q24 hr.
- ***Corticosteroids*** are indicated in inflammatory liver disease; additional immunosuppressive agents may be necessary.
- ***Colchicine*** is antifibrotic; its efficacy is unproven; it can have significant side-effects.
- ***Lactulose*** is used in hepatic encephalopathy as it reduces colonic bacterial numbers and converts NH_3 to NH_4^+ in the colon that is not absorbed. 0.5–1 ml/kg PO q8 hr to give soft stool consistency. Use as an enema in acute encephalopathic crisis, 20 ml/kg (30% solution).
- ***Ursodeoxycholic acid*** **(UDCA)** is choleretic and anti-inflammatory.
- ***Diuretics*** may help in ascitic cases: give spironolactone (2–4 mg/kg q24 hr).
- ***Zinc*** is antifibrotic and decoppering: give 1–2 mg/kg elemental zinc PO q24 hr.
- ***D-penicillamine and trientine*** are decoppering agents (consult appropriate text for use, adverse reactions and drug interactions).

NUTRITION

3.5.9 DIETARY APPROACH TO REDUCED LIVER FUNCTION

- Aim to meet necessary nutritional requirements during times of hepatic insufficiency.
- Malnutrition is common in chronic hepatic disease and adversely affects cellular function, worsening the disease process.
  - It is associated with anorexia, impaired nutrient digestion and absorption, increased energy requirements, and accelerated protein breakdown in conjunction with reduced synthesis.
- Significantly less is known about the nutritional requirements of cats with liver disease than dogs.
- ***Control of ammonia production***
  - Ammonia is produced by protein catabolism and bacterial fermentation.
  - 50% of whole-body ammonia uptake occurs in the muscle.
  - Protein restriction is only appropriate when protein intolerance has developed, but subtle encephalopathy is difficult to diagnose.

  - Dietary proteins should be moderately restricted and of high biological value, limiting nitrogenous waste while maintaining positive nitrogen balance to prevent muscle breakdown.
  - Frequent small meals are beneficial.
  - Protein restriction in puppies/kittens can cause critically low blood albumin; standard hepatic diets are inappropriate and if used should be supplemented: milk or soya-based protein, e.g. cottage cheese. Increase protein to ≈20% of calories fed and monitor clinical signs and albumin.
- ***Fat***: should be restricted to 25–30% of calories.
- ***Carbohydrates***: complex carbohydrates as major energy source.
- ***Fibre*** decreases ammonia production and colonic transit time.
- ***Vitamins***: requirement is increased, as liver is a major site of synthesis, storage, and conversion. Vitamin E scavenges free radicals.
- ***Copper***: should be restricted, as it accumulates due to cholestasis or copper storage diseases. Consider copper content in water: this can be high.
- ***Zinc***: supplement (see p.**), as absorption is decreased and loss increased. Zinc acts to decrease copper absorption and aid ammonia detoxification in the urea cycle. May have
- antifibrotic activities.
- ***Amino acids***: L-carnitine, taurine, glutamine, arginine.
- ***Nutritional supplements***: S-adenosyl methionine, silybin.

SELECTED TOPIC

3.5.10 PORTOSYSTEMIC SHUNTS

- A vascular anomaly resulting in blood bypassing the liver.
- They can be intrahepatic (large-breed dogs) or extrahepatic (small dogs and cats). Extrahepatic shunts are most commonly from portal or mesenteric vein to vena cava or azygous vein.
- Can be clinically silent.
- Presentation is usually poor growth and appetite, with signs of severe liver dysfunction (see 3.5.4). Kittens often have copper-coloured irises.
- Diagnosis requires CT, skilled ultrasonography, or portovenography for confirmation.
- Management (surgical vs medical) remains controversial.
- Acquired shunting will occur if there is portal hypertension.

3.5.11 GALL BLADDER DISEASE

- Gall bladder (GB) disease seems to be becoming significantly more common in dogs. This may reflect changes in breed prevalence.
- ***Acute biliary obstruction***: can be partial or total associated inspissated bile, liths, hepatocyte swelling collapsing intra-hepatic canaliculae, pancreatic disease, neoplasia.
- ***Choleliths and choledocholiths***: composed of precipitated bile compounds and/or calcium. Calcium carbonate liths (radiodense) rare in dogs, common in cats. Cause inflammation, obstruction, and rupture. Imaging to diagnose; surgery required if significant disease.
- ***Gall bladder mucocoele***: bile-laden semisolid mucoid material integrated into wall. Can cause chronic infection and pain, eventually GB rupture and bile peritonitis.
  - Some breeds predisposed, e.g. Shetland sheepdog and Border terrier. Early stages may respond to high dose UDCA; later stages require cholecystectomy.
- ***Neoplasia***: cystadenoma and carcinomas – rare

3.5.12 LIVER NEOPLASIA

- Frequent organ for metastatic disease, but also primary neoplasia.
- Clinical signs often vague, lethargy, inappetence, weight loss, GIT signs, PU/PD, pyrexia, and ascites. Half of cats and a quarter of dogs show no signs at diagnosis.
- Liver enzymes may not be that increased.
- Primary tumours more likely to cause hypoproteinaemia and hypoglycaemia.
- Abdominal ultrasound or CT most likely to show presence of a mass but not all masses are neoplastic.
- Diagnosis on FNAB, needle core or surgical biopsy.
- Treatment – surgical excision, if possible, (chemo)embolisation if not resectable. Poor response to radiation or chemotherapy for primary tumours.
- Most common metastatic/multiorgan tumours – carcinomas, lymphoma, haemangiosarcoma, histiocytic disease, mast cell.
- Most common primary tumours:
    - Solitary hepatocellular carcinoma.
    - Nodular or diffuse hepatocellular carcinoma.

3.6 SPLEEN

3.6.1 *Ultrasound-identified lesions of the spleen* 3.6.2 *Splenic neoplasia* 3.6.3 *Splenic rupture*

INTRODUCTION

- The spleen is a major extramedullary haematopoietic organ. In dogs it is relatively large and contractile; in cats the spleen is small and non-contractile. The spleen is most easily investigated with ultrasound and fine needle aspirate. Usually has a homogeneous parenchymal texture. Changes in echogenicity can be pathologic but may reflect extramedullary haematopoiesis (EMH).

3.6.1 ULTRASOUND-IDENTIFIED LESIONS OF THE SPLEEN

- Small, often hypoechoic, lesions of the spleen are frequently identified on routine abdominal ultrasound in dogs.
- Diagnosis based on ultrasound appearance is not possible the vast majority are not neoplastic but represent EMH or localised inflammation (splenitis). FNAB can be undertaken if the lesion(s) are of sufficient size. Monitoring with serial ultrasound is appropriate. Hyperechoic lesions are also seen and are usually myelolipomas.

3.6.2 SPLENIC NEOPLASIA

- Neoplasia is rare in cats.
- In dogs haemangiosarcoma (HSA) is the most common neoplasm usually presenting either as a palpable mass, lethargy, weakness, and decreased appetite or collapse.
    - Ultrasound will usually establish a rapid diagnosis of a mass ± haemoabdomen appearance can vary from hyperechoic to anechoic; lesions are usually cavitated with hypoechoic blood-filled spaces.
    - Splenectomy is treatment of choice to confirm diagnosis. Median survival times are short (weeks) in HSA cases; life expectancy can be extended with adjunctive chemotherapy.
- Staging should be carried out as HSA in other locations is common especially liver, heart, and lungs.

- Differentials would include haemangioma and haematoma.
- Lymphoma and mast cell tumours are the next most common neoplasms.

3.6.3 SPLENIC RUPTURE

- The most common cause of haemoabdomen in the dog rupture is most commonly associated with trauma or tumours. Signs of tumour rupture can be relatively non-specific with the patient showing abdominal discomfort and brief weakness or collapse (as the splenic capsule tears) and then apparent recovery; rupture often occurs at exercise. Prognosis depends on cause and speed of diagnosis.

3.7 KIDNEYS

3.7.1 *Role of the kidney* 3.7.2 *Risk factors and signs of acute kidney injury* 3.7.3 *Clinical signs of chronic kidney disease* 3.7.4 *IRIS classification* 3.7.5 *Renal imaging* 3.7.6 *Therapy for chronic kidney disease* 3.7.7 *Dietary management of chronic renal disease* 3.7.8 *Diagnosis of acute kidney injury* 3.7.9 *Management of acute kidney injury*

INTRODUCTION

3.7.1 ROLE OF THE KIDNEY

- The main functions of the kidney are:
  - Excretion.
  - Water balance.
  - Blood pressure.
  - Electrolyte balance.
  - Acid/base status.
  - Hormones:
    - Excretion, e.g. gastrin.
    - Production of erythropoietin, calcitriol, and renin.
    - Conversion of vitamin D.
- The basic functional unit of the kidney is the nephron (Figure 3.7 and Table 3.7a).
- Chronic disease leads to progressive loss of nephrons, with hyperfiltration occurring in the remainder to compensate. Eventually a tipping point occurs, where the level of hyperfiltration causes further nephron loss regardless of initiating cause, resulting in disease progression.

HISTORY AND PHYSICAL EXAMINATION

3.7.2 RISK FACTORS AND SIGNS OF ACUTE KIDNEY INJURY

- Risk factors are additive and include pre-existing renal disease, sepsis, muscular damage, diabetes, hypovolaemia, electrolyte and acid/base imbalance, nephrotoxic drugs, and low protein diets.
- Clinically these cases present with non-specific signs of lethargy, depression, anorexia (2.4), and collapse (5.6).
- Owners may not have noticed changes in urine production.
- Cases will occur in hospitalised patients. It is therefore essential to flag a lack of urine production as soon as possible, to allow aggressive early intervention.

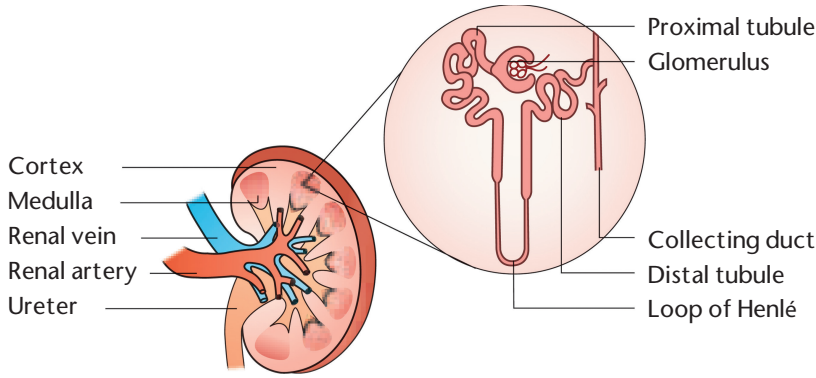


Figure 3.7 Anatomy of the kidney and nephron.

Table 3.7a Nephron functions	
Nephron region	**Function**
Glomerulus	Filtration, renin production, plasma volume regulation
Proximal tubule	Resorption of glucose, proteins, amino acids, vitamins, electrolytes; secretion of H^+
Loop of Henlé	Counter-current concentration of Na^+ and urea in the medulla
Distal tubule	Secretion of H^+ and K^+ ions
Collecting duct	Selective permeability to water influenced by antidiuretic hormone

3.7.3 CLINICAL SIGNS OF CHRONIC KIDNEY DISEASE

- Commonly: polyuria/polydypsia (2.8), weight loss (2.7), lethargy, poor appetite (2.4), depression, poor hair/coat.
- Rarely: vomiting (2.5), diarrhoea (2.6), blindness.

DIAGNOSIS

3.7.4 INTERNATIONAL RENAL INTEREST SOCIETY (IRIS) CLASSIFICATION

- Chronic renal disease should be fully evaluated, as this will be part of the patient's physiology for the rest of their life.
- IRIS publishes guidelines (2023), including disease stage.
- Classification (Table 3.7b). Staging facilitates appropriate treatment and monitoring (3.6.6).
- Note that creatinine value is irrespective of laboratory reference range, as animals in the upper reference interval often have excretory failure and this extends into the mid-range in very thin patients. Symmetric dimethyl arginine (SDMA) level is also included in the grading system
- IRIS staging is subdivided according to:
  - Proteinuria, classified as non-proteinuric (NP), borderline proteinuric (BP), and proteinuric (P).
  - Persistent hypertension (Table 3.7c).
  - Evidence of target organ damage (TOD) damage from hypertension (3.3.87):
    - nc: no complications.
    - c: complications (TOD).
    - rnd: risk not determined.

Table 3.7b IRIS classification of CKD

Stage	**Plasma creatinine** (μmol/L) **SDMA** (μg/dL) *Dog	Cat	**Comments**
1	<125 <18	<140 <18	Non-azotaemic. Kidney abnormal based on urine concentration, palpation, proteinuria or biopsy
2	125–179 18–35	140–249 18–25	Mild renal azotaemia; clinical signs usually mild or absent
3	180–439 36–54	250–439 26–38	Moderate renal azotaemia; many systemic clinical signs may be present
4	>440 >54	>440 >38	Severe renal azotaemia; many extra-renal signs present

* average size

Table 3.7c IRIS substaging by arterial blood pressure

Systolic BP (mmHg)	Breed specific reference range (RR)	Substage risk
<150	<10 mmHg above RR	Minimal (N)
150–159	10–20 mmHg above RR	Low (L)
160–179	20–40 mmHg above RR	Moderate (M)
≥180	≥40 mmHg above RR	High (H)

  - So, a dog with a creatinine of 160 μmol/l, a UPC of 0.4 and a systolic BP of 155 mmHg, with no evidence of end-organ damage, would be classified as IRIS stage 2–BP–L–nc.
  - Stage reclassified if treatment of BP or proteinuria .
- Beyond measuring creatinine, urea, and blood pressure (3.3.8), management can be improved with knowledge of red cell count, electrolytes, calcium, phosphate, albumin, and full urinalysis (1.3.6). IRIS sets specific targets for phosphate at different stages.

3.7.5 RENAL IMAGING

- Radiographs can be used to determine renal size and shape and the presence of pelvic mineralisation/uroliths, as well as ureteroliths.
  - Normal renal size can be compared with the size of lumbar vertebra L2 and the aorta (Table 3.7d).
- Ultrasound is more sensitive for renal shape and architecture, including pelvic dilation, as well as variation within and differentiation between the medulla and cortex, e.g. cortical cysts. With experience, ureteric dilation can also be evaluated.

3.7.6 THERAPY FOR CHRONIC RENAL DISEASE

- Therapy is based on the IRIS staging system as follows:
  - ***Stage 1***
    - Discontinue any nephrotoxic drugs and rule out treatable underlying disease, e.g. pyelonephritis.
    - Correct dehydration and other pre- and post-renal causes.
    - Treat hypertension as per guidelines (3.3.8).
    - Investigate proteinuria, if present; consider angiotensin-converting enzyme inhibitor (ACEi)/angiotensin-receptor blocker (ARB) if UPC >0.5 dog or >0.4 (cat) and clopidogrel or aspirin if albumin <20g/l.

Table 3.7d Reference ranges for renal size

Imaging mode	Normal renal size	
	Dog	Cat
Radiography	2.5–3.5 x length L2	2.4–3.0 x length L2
Ultrasound	Ratio of kidney length to aorta diameter: 5.5–9.1	Length: 27–47 mm (35 av.) Width: 17–28 mm (21 av.)

L2 = second lumbar vertebra

- ***Stage 2***: As stage 1, plus
  - Increased UPC confirmed and repeatable should be treated with ACEi or ARB; combination therapy may be necessary
  - Manage phosphate levels to between 0.9 and 1.45 mmol/l, by diet, phosphate binders given with each meal.
  - Treat acidosis if present (sodium bicarbonate/potassium citrate).
  - Potassium gluconate if hypokalaemic – usually cats.
  - Where creatinine suggests stage 2 but SDMA stage 3 treat as stage 3
- ***Stage 3***: As stage 2, plus
  - Target phosphate below 1.5 mmol/l (dog) 1.6 mmol/l (cat).
  - Treat anaemia if affecting quality of life (erythropoietin, hypoxia inducible factor [molidustat] transfusion).
  - Manage nausea/vomiting (antacids, mucosal protectants, antiemetics).
  - Maintain hydration with parenteral fluids if necessary.
  - Consider treating anaemia if affecting QoL.
  - Where creatinine suggests stage 3 but SDMA stage 4 treat as stage 4.
- ***Stage 4***: As stage 3, plus
  - Target phosphate below 1.9 mmol/l.
  - Avoid drugs requiring renal clearance or adjust dose.
  - Try to maintain body weight, lean body mass nitrogen balance and hydration (appetite stimulants, e.g. mirtazapine, gherlin agonist [capromorelin], subcutaneous fluid).
- If necessary consider fitting a percutaneous endoscopic gastrostomy (PEG) tube.
  - Antimicrobials should be given if there is a positive urine culture, or evidence to support pyelonephritis (3.13.5).

! Take care to ensure patient is normally hydrated when introducing anti-hypertensive, ARB or ACEi therapy, especially in stage 4 disease.

NUTRITION

3.7.7 DIETARY MANAGEMENT OF CHRONIC KIDNEY DISEASE

- Diet is an essential part of the management of chronic kidney disease (CKD), more than doubling the median life expectancy in cats and dogs.
- Not all cats and dogs will tolerate the ideal dietary modification so compromises may need to be made. It is vital that the patient eats something, as body protein catabolism will have more serious adverse effects than almost any diet.
- Aims:
  - Reduction in hyperphosphataemia and azotaemia.
  - Reduction in associated nausea, vomiting, and anaemia.

  - Improvement in body condition and water-soluble vitamin status.
  - Correction of hydration, acid/base balance, and electrolytes.
  - Reduction in blood pressure via omega-3 fatty acids and sodium balance.
  - When to start: the ideal time is not known, but definitely when hyperphosphataemia develops and possibly when there is significant azotaemia (urea >12–13 mmol/l, creatinine >180 µmol/l (dog) 250 µmol/l (cat) even without elevated phosphate).
- Diet choice: highly palatable, wet food is preferable.
  - Protein restricted: 14–15% (dog); 28–30% (cat) protein on a dry matter (DM) basis.
  - Phosphate restricted: 0.15–0.3% (dog); 0.4–0.6% (cat) on a DM basis.
  - Potassium and water-soluble vitamin supplemented.
  - Soluble fibre up to 10% DM helps to trap nitrogen.
  - Increased omega-3 polyunsaturated fatty acids may help.
  - Non-acidifying with reduced salt (NaCl) levels.
  - Energy from fat as it increases density and palatability.
- Fluid balance: a crucial element of therapy is aimed at maximising oral fluid intake. If this is inadequate, then subcutaneous fluid can be considered.

! Always check whether a patient is eating normally before interpreting serial changes in urea and creatinine. Starvation will result in a fall in urea and creatinine levels.

SELECTED TOPICS

3.7.8 DIAGNOSIS OF ACUTE KIDNEY INJURY

- This is signalled by a sudden rise in creatinine by >50%.
- Prompt intervention is crucial to interrupt the induction/extension phase and limit nephron loss.
- Be alert to risk factors – nephrotoxic drugs commonly NSAIDs pre-existing renal, heart, pancreatic disease, geriatric, sepsis, fever, systemic disease, e.g. vasculitis, neoplasia, GDV, leptospirosis, muscular damage – e.g. trauma, diabetes mellitus, hypoalbuminaemia, electrolyte, and/or acid/base imbalance, recent anaesthesia, hypovolaemia, hyperviscous states.
- Diagnosis is via physical examination, risk factors and speed of onset of signs.
  - Note: kidneys often look normal on ultrasound.
  - Magnitude of azotaemia may not be especially extreme for the level of clinical signs, and often worsens despite treatment.
  - Patients are usually proteinuric, with alkaline urine. They can be anuric to polyuric.
  - Renal biopsy may help, but it is technically complicated.

3.7.9 MANAGEMENT OF ACUTE RENAL DISEASE

Treatment goals

- Prevent further renal damage: induce emesis, use gastrointestinal adsorbents, and stop nephrotoxic drugs.
- Maintain renal perfusion.
- Maintain urine flow and limit rise in uraemic toxins by diuresis using fluid therapy, furosemide, mannitol.
  - Note: increased urine flow alone does not mean improved glomerular filtration rate (GFR)

- Correct acid/base and electrolyte abnormalities.
  - Note: electrolyte abnormalities are often associated with cellular shifts rather than absolute change, so they can alter very rapidly.
  - Primary correction of acidosis is rarely required.
- Control uraemic damage to GIT: use antacids and sucralfate.
- Control vomiting: use metoclopramide (not with dopamine CRI), maropitant, prochlorperazine, dolasetron, ondansetron.
- Provide antibacterial cover, as uraemic patients are immunocompromised.
- Peritoneal dialysis is possible, but technically challenging.
- Haemodialysis is available in some countries.

Treatment tips

- Always place a urinary catheter and measure output 1–2× hourly.
- Fluids are the cornerstone of treatment.
- If there is oliguria or anuria, avoid over-hydration, particularly in cats.
- Safe volume expansion is 5% once hydration status is normal.
- An 'in-and-out' approach can be used: measure urine output/hr, add 1 ml/kg to that figure, and replace that amount in the following hour.
- Failure to re-establish urine output within 6–12 hours carries a guarded prognosis.

3.8 LOWER URINARY TRACT

3.8.1 *Disease types and bladder innervation* 3.8.2 *Differentiating disease types* 3.8.3 *Diagnostic tests* 3.8.4 *Drug therapy* 3.8.5 *Dietary management of LUT disease* 3.8.6 *Prostatic disease*

INTRODUCTION

3.8.1 DISEASE TYPES AND BLADDER INNERVATION

- Lower urinary tract (LUT) diseases fall into three main groups:
  - cystitis (pollakiuria) (2.9), incontinence (2.10), and obstruction (5.7). For urinary catheterisation, see 1.4.15a and 1.4.15b.
- Neurologic control of bladder function is mediated by the hypogastric, pudendal, and pelvic nerves (see Figure 3.8).
- Spinal cord damage may affect bladder function, depending on its location. Damage above L4–L5 tends to cause a spastic bladder and inability to urinate, while damage to the spine below L4–L5 causes a flaccid overflow bladder.

HISTORY AND PHYSICAL EXAMINATION

3.8.2 DIFFERENTIATING DISEASE TYPES

- In the case of frequent attempts to urinate, initial examination should aim to differentiate cystitis from obstruction.
  - Cystitis patients tend to be restless, with frequent attempts to urinate – squatting and straining, often with haematuria but are rarely systemically unwell.
  - Obstructed patients tend initially to make frequent urination attempts and then will often appear to settle down, but they become quieter and more withdrawn as the metabolic changes associated with obstruction take effect.

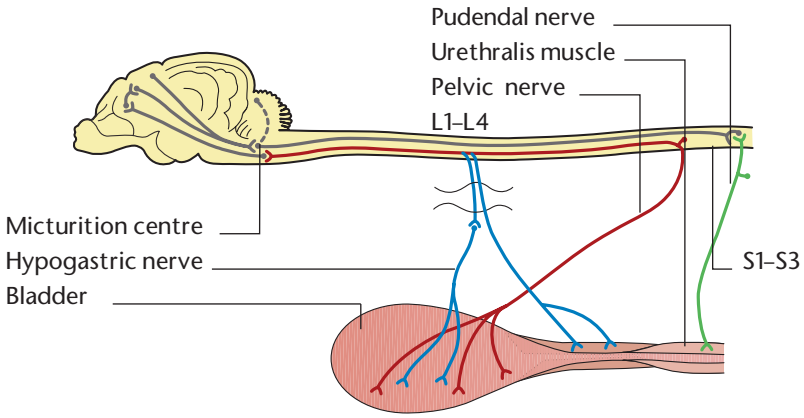


Figure 3.8 Innervation of the bladder.

- Incontinence should be distinguished from increased urination associated with PU.
- In chronic feline idiopathic cystitis (FIC) an in-depth history covering the cat's home environment is essential.

DIAGNOSIS

3.8.3 DIAGNOSTIC TESTS

- In the absence of obstruction or signs of PU/PD, biochemistry, and haematology are rarely of great diagnostic value.
- Full urinalysis (including UPCR and culture) is an essential early diagnostic test.
  - Ideally perform culture on a cystocentesis sample. If free-catch then at least 10^5 CFU/ml in a midstream sample would suggest infection especially if culture monomorphic and the sediment contains increased white and epithelial cells.
- Crystals in urine sediment do not necessarily indicate:
  - That crystals are present in the bladder, unless it is a fresh sample placed on a warmed slide.
  - That any uroliths present are of the same composition.

IMAGING

- Radiographic evaluation of the lower urinary tract can be achieved using:
  - Plain radiographs, with double contrast cystography and retrograde urethrography, to look at bladder size and position, wall thickness and regularity, filling defects, presence of uroliths, and urethral stricture.
  - Intravenous urography to determine ureteral patency and positioning.
- Ultrasound can be used to evaluate all of these areas, apart from normal ureters, and much of the male and the distal female urethra. It has the advantages of being quicker, safer, less expensive, and does not require anaesthesia.

RADIODENSITY AND APPEARANCE OF UROLITHS

- Uroliths vary in radiodensity. Diagnosis requires both plain and contrast radiographs.
- Air bubbles introduced with the contrast medium can mimic uroliths. To distinguish the two: bubbles rise, uroliths sink under gravity.

TREATMENT

3.8.4 DRUG THERAPY

- Appropriate therapy for lower urinary tract disease is dependent on accurate diagnosis (Table 3.8b and algorithm 2.9). Evidence for drug efficacy in the management of recurrent cyctitis and urethrospasm is poor.

Treatment of urinary tract infection

- Urinary tract infection (UTI) is divided into occult, simple, and complex infections.
- Occult infections (no clinical signs or urinary change) not normally treated.
- Many cats with urinary signs are treated with antimicrobials when no infection is present – ideally, based on culture and sensitivity using narrowest spectrum antimicrobial.
    - ***Simple*** duration is controversial move generally to shorter course of 1–3 days. Amoxicillin or potentiated sulphonamide usually effective.
    - ***Complex*** associated with predisposing cause can be reinfection, relapse or refractory. Manage predisposing cause as far as possible. Longer antimicrobial course may be necessary. Re-culture during and after course advised.

NUTRITION

3.8.5 DIETARY MANAGEMENT OF LUT DISEASE

- For idiopathic cystitis or urolithiasis, diet should be the first-choice therapy, as it is safer and may be more effective than drugs or surgery (Table 3.8a).
- ***Crystalluria***: if asymptomatic, dietary intervention is not indicated.
- ***Idiopathic cystitis***: diets which increase urine output.
- ***Urolithiasis***: see Table 3.8b
- A wide variety of urinary diets are available. Some are marketed to promote 'urinary health' other have specific claims about prevention or dissolution. Choose according to intended purpose.

Table 3.8a Characteristics of diets for urolith management

	Struvite (dissolution)	Struvite (prevention)	Calcium oxalate (prevention)	Urate (dissolution & prevention)
Protein	<8% DM	<25% DM	10–18% DM	10–18% DM
Calcium			0.3–0.6% DM	
Magnesium	0.02% DM	0.04–0.1% DM	0.04–0.15% DM	
Phosphorus	<0.1% DM	<0.6% DM		
Oxalate			Avoid high oxalate foods	
Vitamin D			500–1500 iU/kg DM	
Vitamin B_6			>1.0mg/kg DM	
Vitamin C dissolution			No supplemented foods or treats	
Target pH morning urine	<6.5	<6.7	Any	7.1–7.7

Table 3.8b Common drug treatment of LUT disease

Problem	Generic drug	Oral dose (mg)	Side-effects
Incontinence	*Phenylpropanolamine (S)	1.0/kg q8hr or 1.5/kg q12hr	Hypertension, restlessness, agression
	*Oestriol (T)	1/dog q24hr	Oestrogenic signs
†Reflex dyssynergia or urethrospasm post obstruction	Diazepam (T)	2–10/dog q8hr 1.25–5/cat q8hr	Sedation, ataxia, muscle weakness, hepatic necrosis (cat)
	Phenoxybenzamine (C)	0.25–1/kg (dog) q8–24hr; 0.5–1/kg (cat) q12hr	Hypotension, tachycardia
	Prazosin (T)	1/15 kg (dog) q8–24hr; 0.25–0.5/cat q12–24hr	Hypotension, sedation, heart failure in occult disease
	Dantrolene	0.5–2/kg q12hr	Muscle weakness, hepatitis, pleural effusion
Cystitis	Consider glycosaminoglycan supplements, increasing water turnover (wet diet, frequency of urination), reducing environmental stressors (cats), amitriptylline		
Urolithiasis	In conjunction with diet for certain uroliths, e.g. allopurinol (urate), D-penicillamine (cystine)		
Urinary tract infection	Should ideally be based on culture and sensitivity (see 3.8.4)		
Bladder neoplasia	Transitional cell carcinomas most common tumour. Some respond well to COX-2 inhibitors (piroxicam, meloxicam). Variable response to chemotherapy (carboplatin, mitoxantrone)		

* Licensed for dogs; T = tablet; S = syrup; C = capsule; † two drugs usually combined to maximize muscle relaxation

SELECTED TOPICS

3.8.6 PROSTATIC DISEASE

- Very rare in cats and uncommon in castrated male dogs. Very common in entire male dogs. Prostatic abscessation and cysts are also seen.
  - ***Benign prostatic hyperplasia***: prostatic size increases under influence of dihydrotestosterone. Prostate becomes symmetrically enlarged but non-painful. Cysts can cause asymmetry. Mostly asymptomatic but if large enough can cause dyschezia and constipation.
  - ***Prostatitis***: can present as acute or chronic disease; usually entire dogs
  - ***Acute*** – typically patients systemically unwell with fever as well as organ-specific signs including stranguria and tenesmus. Prostate on palpation asymmetric and painful often very painful to the point that even a well-behaved dog may try and bite. Confirmation with ultrasound and prostatic/urine culture. Intensive treatment may be required.
  - ***Chronic*** – bacterial disease more common than in acute cases. Usually presents as recurrent UTI, haematuria, bleeding independent of urination, pain. Prostate usually not painful. Diagnosis on imaging and prostatic wash.

- ***Prostatic neoplasia***: usually adenocarcinoma, can arise from a variety of different structures including urethra. Present with dysuria, obstruction, haematuria, dyschezia, hind limb pain, and ataxia. Diagnosis on history, imaging and cytology/histology. Local lymphadenomegaly common can spread to lungs and bone. Survival times can be short. Can consider COX-2 inhibitors with carboplatin. If the patient is obstructed, then cystotomy tube or stenting the urethra can be considered.

3.9 REPRODUCTION

3.9.1 *Presentation* 3.9.2 *Vital history questions* 3.9.3 *Routine tests* 3.9.4 *Monitoring the oestrous cycle in bitches* 3.9.5 *Pseudopregnancy* 3.9.6 *Mismating or unwanted pregnancy* 3.9.7 *Vaginal discharge and pyometra* 3.9.8 *Phimosis and paraphimosis* 3.9.9 *Caesarean section* 3.9.10 *Post-parturient conditions* 3.9.11 *Dietary management* 3.9.12 *Neonatology*

INTRODUCTION

3.9.1 PRESENTATION

- The majority of cats and dogs present for routine neutering to control fertility. (WSAVA guidelines for the control of reproduction in dogs and cats. Journal of small animal practice (2024) 65 pg 424–559)
    - In dogs, neutering reduces risks of some diseases but increases the risks of other. Relative risk varies with breed. Owners may want to discuss the pros and cons of neutering.
- Presentation for reproductive problems is relatively rare in cats other than for medication to postpone oestrus.
- Common presentations in dogs would include pseudopregnancy, pyometra, and juvenile vaginitis.
- Apart from infertility and closed pyometra, the starting point for most reproductive problems is self-evident on history or physical presentation.
- Infertility frequently presents without any outward signs of ill health and can be daunting when dealing with valuable patients and breeders with considerable knowledge and experience. Knowing what can represent normal variation in the female reproductive cycle is a useful starting point.
    - For an approach to infertility see Figure 3.9. Inevitably, some mating will fail without evidence of clear cause, despite extensive investigation.

Table 3.9a Female reproductive parameters

	Dog	Cat
Age of reproductive maturity	6–12 months	5–12 months
Oestrous cycle (frequency)	3.5–13 (6) months	Induced ovulation
Duration of oestrous cycle	7–35 (16) days	
Gestation period	56–72 (63) days	56–71 (63) days
Litter size	2–15	3–9 (4)
Earliest age of weaning	4 weeks	4 weeks

Parameters vary with breed; figure in parentheses is the average

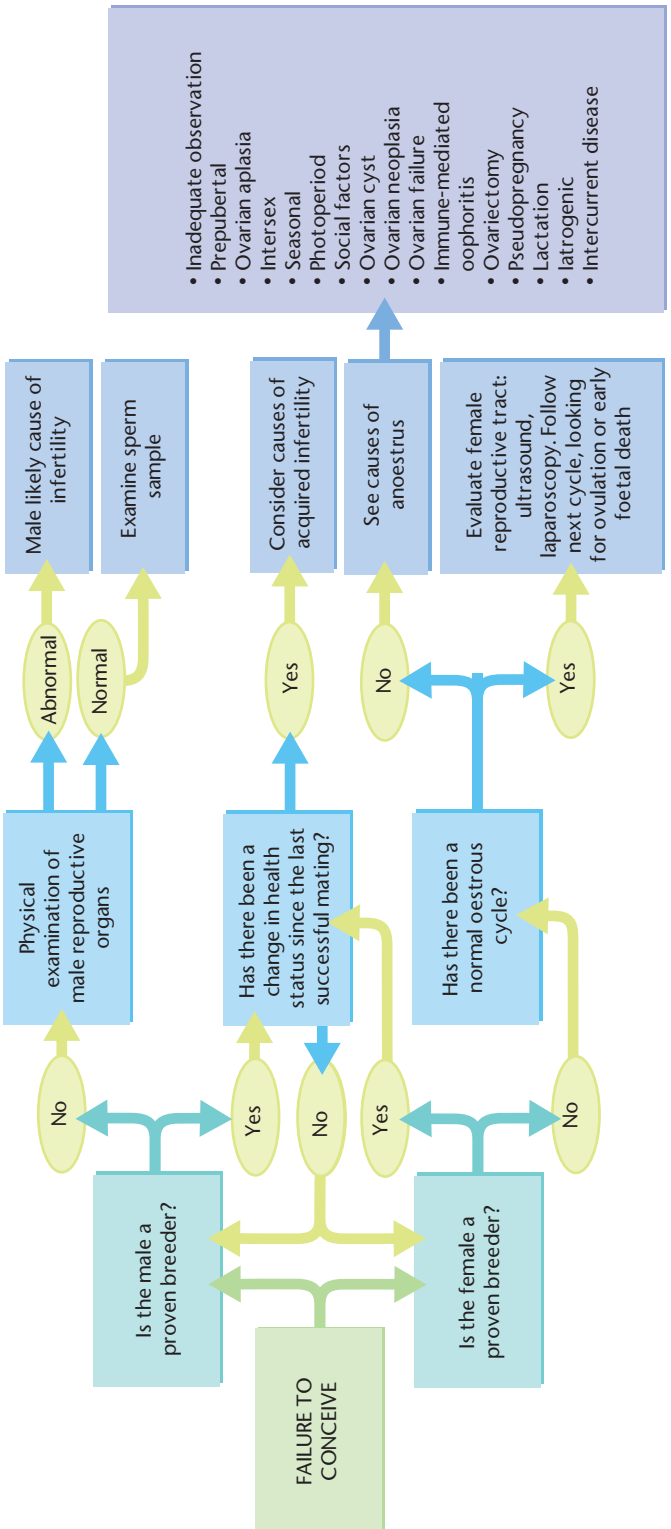


Figure 3.9 Algorithm illustrating an approach to infertility.

HISTORY AND PHYSICAL EXAMINATION

3.9.2 KEY HISTORY QUESTIONS

- Neutering status should always be checked, as mistakes in records are relatively common.
- In intact patients, a full reproductive history is vital, as the approach to patients that have previously mated successfully is different to those that have not.
- It is assumed that patients that have previously mated successfully do not have hereditary or congenital anatomic or genetic abnormalities that would prevent conception.
- However, they may have acquired disease, e.g. endometritis; be incompatible with their chosen mate; or have developed issues with their reproductive cycle.
- In bitches, a description of their cycling frequency and length, and behaviour during oestrus should be obtained.

DIAGNOSIS

3.9.3 ROUTINE TESTS

- Routine haematology and biochemistry in reproductive disease help to rule out intercurrent disease and pyometra.
- The state of the uterus and ovaries can be evaluated using ultrasound.

3.9.4 MONITORING THE OESTROUS CYCLE IN BITCHES

- The stage of the oestrous cycle can be monitored by plasma progesterone, or by vaginal cytology.
- Plasma progesterone (see Table 3.9c):
  - If rise from <1 ng/ml to 2–4 ng/ml, ovulation in about 2 days (increased frequency of samples increases accuracy).
  - 4–10 ng/ml indicates ovulation.
  - 10–25 ng/ml indicates fertilisation.
- Vaginal cytology: see Table 3.9b. See also individual conditions (3.9.5–3.9.7).

COMMON REPRODUCTIVE PROBLEMS

3.9.5 PSEUDOPREGNANCY (FALSE PREGNANCY)

- Cats: can follow sterile mating, spontaneous ovulation or early embryonic death, leading to 40–50 days without oestrus.
- Dogs: common 6–14 weeks following oestrus.
  - Common signs are mammary development, usually with milk, aggression, dullness, nesting/toy fixation, inappetence, vulval discharge (milky).
- Treatment unnecessary if signs are mild.
  - Lactation can be suppressed e.g. by preventing the bitch from licking her mammary glands, exercising to reduce nesting behaviour.

Table 3.9b Vaginal cytology

	Pro-oestrus		Oestrus				
Cell type	**Early**	**Late**	**Early**	**Late**	**Early metoestrus**	**Anoestrus**	**Vaginitis/ pyometra**
Parabasal	+++	+	–	–	–	+	–
Intermediate	+++	++	–	–	+	+	–
Superficial	+/–		+++	+++	–	–	–
RBCC	+	+++	++	–	–	–	–
Neutrophils	+	–	–	+	+++	+	++++

Table 3.9c Progesterone levels

	Pro-oestrus	LH surge	Ovulation	Fertilization
Progesterone (ng/ml)	<1	2–4*	4–10	10–25

* ovulation around 2 days later

- For more severely affected bitches, use prolactin inhibitors: cabergoline (5 µg/kg PO q24 hr for 5–10 days) suppresses lactation in 95% of cases.

3.9.6 MISMATING OR UNWANTED PREGNANCY

- ***Treatment***: a variety of regimes are described; aglepristone is licensed in the UK. Other drugs such as cabergoline, prostaglandin (PG) F2α and oestrogens can also be effective.
  - Potential complications should be discussed with owners prior to treatment.

3.9.7 VAGINAL DISCHARGE AND PYOMETRA

- ***Vaginitis***: patient is systemically well, haematology within normal limits. Juvenile vaginitis from 2–3 months old usually resolves with the first season. Adult vaginitis should prompt investigation of underlying cause.
- ***Pyometra***: is a consequence of cystic endometrial hyperplasia allowing secondary infection during oestrus. Can occur in spayed bitches as a stump pyometra usually means an ovarian remnant is present.
  - Signs of pyometra are usually apparent 6–8 weeks post-season. If pyometra is causing systemic toxicity, patients are typically dull, inappetent, with PU/PD. Vaginal discharge may be present.
- Confirm with haematology and imaging (ultrasound).
- Treatment: for most patients stabilisation and ovariohysterectomy is the best choice.
- Medical management can be considered for reproductively valuable individuals. Aglepristone, dopamine agonists and PGF2α agonists have been used.

3.9.8 PHIMOSIS AND PARAPHIMOSIS

- ***Phimosis***: inability to extrude penis; can cause dribbling urination or infertility. Management is surgical.
- ***Paraphimosis***: an engorged or oedematous penis that cannot be retracted. Obstruction of circulation to the penis will lead to desiccation, ischemia, urethral obstruction and gangrene.
  - Paraphimosis is most commonly due to post-coital hair entanglement – a medical emergency.
  - Treatment: Cleansing, sedation, lubricant and cold hypertonic dextrose applied to penis; surgical enlargement of preputial orifice and indwelling urinary catheter; ultimately amputation.

3.9.9 CAESAREAN SECTION

- Indications:
  - Primary or secondary uterine inertia.
  - Abnormality of maternal pelvis or soft tissue of birth canal.
  - Relative/absolute foetal oversize or deficient/excess foetal fluid.
  - Foetal death with putrefaction, toxaemia or other maternal illness.
  - Dystocia due to malposition or neglect.
  - Previous history of prophylaxis, or breed-specific prophylaxis, e.g. bulldog.
- Anaesthesia has increased issues due to:
  - Reduced maternal respiratory capacity.
  - Increased sensitivity of respiratory centre to CO_2.
  - Acid/base disturbances (variable).

  - Blood and fluid loss at surgery.
  - Partial vena cava occlusion.
  - Foetal depression caused by anaesthetic agents.
- Suitable anaesthetic regimes: a variety are appropriate; pre-oxygenation of the dam is important. In the majority of cases the practice's standard anaesthetic protocols should be used.
  - Premedication with methadone alone is preferable. If further sedation required consider ACP. Induction with lowest dose of propofol or alfaxalone (potentially safer) to allow intubation with inhalation maintenance.
  - Ketamine/medetomidine as an IM technique with atipamezole (one drop on the tongue) given to the neonate if IV induction is not possible. Causes more respiratory depression and is less controlled.
  - CRI propofol is not recommended.

3.9.10 POST-PARTURIENT CONDITIONS

- Generally, the bitch/queen recovers quickly from vaginal birth or caesarean. If this does not occur, possible causes are:
  - Uterine haemorrhage.
  - Retained placenta(s) or foetus(es).
  - Acute metritis.
  - Uterine rupture or prolapse.
  - Toxic milk syndrome.
  - Acute mastitis.
  - Galactostasis.
  - Eclampsia.
- Many of these are emergencies and require rapid diagnosis and treatment.

NUTRITION

3.9.11 DIETARY MANAGEMENT

- See lifestage nutrition (1.1.9)

SELECTED TOPIC

3.9.12 NEONATOLOGY

Fading puppy/kitten

- Reference ranges can be found elsewhere in this book for neonatal physiology (1.2.6) and for haematology and biochemistry (1.3.1, 1.3.2).
- A fading puppy or kitten is born apparently healthy, but gradually becomes inactive, loses its suckle reflex and may die in the first few weeks of life. Localising signs are usually absent.
- This condition represents a clinical description rather than a diagnosis: it could be due to a multitude of infectious and non-infectious conditions.

Evaluating the neonate

- History: include a breeding history of the household; kennel or cattery management (hygiene, worming, vaccination, and so on); health of the dam during the pregnancy; health of the remainder of the litter; age of the puppy/kitten; and the pattern of the illness to date.
- Death within the first 2 weeks is usually associated with genetic problems, *in utero* issues or trauma.
- Deaths after weaning are most likely infectious, usually viral.

- Clinical examination is difficult. It becomes more rewarding as the patient gets older and begins to develop adult responses. Neonates show limited responses to disease, initially becoming agitated and crying, progressing to inactivity, hypothermia, and loss of the suckling reflex. They deteriorate rapidly with severe infection. Weight gain can be a sensitive indicator of developing problems. Failure to gain weight over any 24-hour period should be investigated.
- Systematic approach to examination is essential.
    - External features: hair/coat (amount, condition, parasites); hydration; signs of injury; umbilicus; dewclaws; (docking site); discharge from nose; urine staining (patent urachus); diarrhoea/rectal patency; congenital malformation.
    - Eyes: swelling under lids indicates pus (often *Staphylococcus* species); eyes open between 5 and 14 (cats) and 10 and 14 (dogs) days, pupillary light response present within 24 hours of the eyes opening, mild corneal cloudiness usually present as eyes open.
    - Ears: external auditory meatus opens between 6 and 14 days; check for mites; middle ear infection (bulging tympanum).
    - Mouth: mucous membrane colour; evidence of cleft palate.
    - Thorax: regular rhythm; heart murmurs may be functional (usually soft); lung sounds difficult to distinguish but should be present; check symmetry/malformation of the thoracic cavity.
    - Abdomen: should feel full, but not swollen or tight; liver and spleen not palpable; intestines soft, mobile and non-painful; urinary bladder freely movable.
    - Neurological assessment: alertness, response to stimulation, suckle reflex; other reflexes appropriate to age; gait (walking from 4 weeks old); posture. Flexor and extensor dominance appears more variable in kittens than puppies.

3.10 SKIN AND EARS

3.10.1 *Skin anatomy and general function* 3.10.2 *General approach to consultation* 3.10.3 *Checklists* 3.10.4 *Diagnostic tests* 3.10.5 *Pruritus* 3.10.6 *Pustules* 3.10.7 *Alopecia* 3.10.8 *Diseases of the claw and claw fold* 3.10.9 *Topical agents* 3.10.10 *Dietary approach to skin disease* 3.10.11 *Otitis*

INTRODUCTION

3.10.1 SKIN ANATOMY AND GENERAL FUNCTION

- A working knowledge of skin anatomy is important in interpreting the results of skin biopsies (Figure 3.10a).
- The skin is the largest organ in the body. It forms an anatomical and physiological barrier between the animal and the environment, along with other functions including:
    - Thermoregulation.
    - Indication of health and reproductive status.
    - Camouflage and UV protection.
    - Immunoregulation and immunosurveillance.
    - Storage of water, fats, vitamins, and other materials.
    - Sensory perception.
    - Vitamin D production.
    - Secretion and excretion.

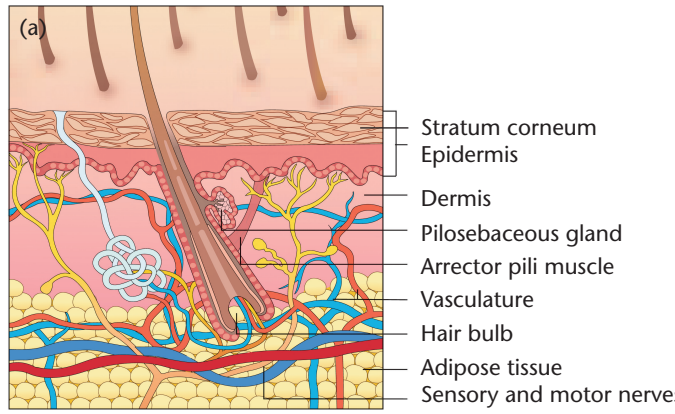


Figure 3.10a Cross section of skin.

HISTORY AND EXAMINATION

3.10.2 GENERAL APPROACH TO CONSULTATION

- Obtain a detailed history.
- Perform a thorough dermatological examination.
- On the basis of the foregoing, construct a differential list for discussion with the owner.
- Agree a course of action, which will lead on to options for further investigation and treatment (3.10.9).

! It is essential to develop a long-term relationship with the owner that encourages open communication and a spirit of cooperation.

- Taking a dermatological history is challenging as the condition may have been present for many years and been treated by multiple clinicians and practices.
- Particularly important are the initial presenting signs reported by the owner and described by the vet, distribution, response to previous treatment, parasite control history, and status of in-contact animals.
- The following link may be useful: https://www.studocu.com/en-gb/document/university-of-surrey/the-veterinary-professional/the-veterinary-professional-consultation-Calgary-Cambridge-model/30204696

3.10.3 CHECKLISTS

Dermatological history

- Presentation: identify presenting complaint and owner's principal concerns.
  - Age of onset, initial clinical signs.
  - Duration of disease, progression of signs, and seasonal variation.
- General history: full general history with special attention to changes in appetite/thirst, gastrointestinal disturbances, sexual status, and history of previous illness or injury.
- Related/in-contact animals and people: parents, littermates, pets belonging to family/friends, casual contacts/dog walker.
  - Is there evidence of zoonotic spread to in-contact people?

ENVIRONMENT

- Indoor: sleeping area/bedding. Access to bedrooms.
- Outdoor: areas visited, swimming, other animals encountered. Check if outdoor access is regulated, on demand or free (cat or dog flap). Nature of surrounding environment.
- Diet: record all foods and liquids offered, occasional extras, leftovers, treats, or bribes given with medication. Record time of meals and whether provided *ad lib* or in set meals.

PAST MANAGEMENT

- Have parasites been seen and how long ago?
- What parasite control measures have been administered, including the frequency and duration of treatment?
- What are the bathing and grooming practices?
- What therapy has been administered for other conditions?
- Prior investigation and treatment: record results and review response to any prior tests, trials or investigations performed.
    - Consider the drugs used and the dose and duration of treatment.

DERMATOLOGIC EXAMINATION

- Record the general appearance, regional distribution, e.g. interdigital (Figure 3.10b), symmetry, nature, e.g. pustular (Figure 3.10c), number, and distribution of clinical lesions. Highlight and explain lesions to owner.
- Check hairs for abnormalities in tip, shaft or root. Check follicles.
- Explain process of examination and solicit owner's input.

DIAGNOSIS

3.10.4 DIAGNOSTIC TESTS

- Routine haematology, biochemistry, hormonal testing, and imaging are appropriate to rule out systemic causes of skin disease.
- Local testing of the skin includes:
    - Wood's lamp for fluorescent fungi (not all *M. canis*).
    - Coat brushing/wet paper test for flea dirt.
    - Hair plucking for damage, stage of the hair cycle, parasites, and follicular plugs.
    - Tape impressions for surface bacteria and yeasts (Figure 3.10d).
    - Scrapes for ectoparasites, bacterial/fungal culture.
    - Cytology aspirates of mass lesions.
    - Skin biopsy of lesions or masses.
    - Allergy testing: intradermal; serologic.

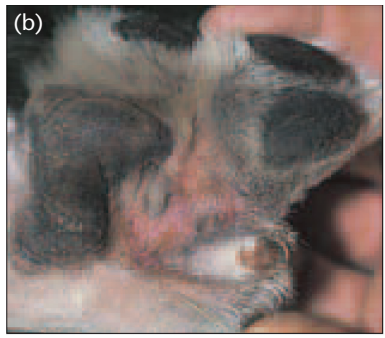
3.10.5 PRURITUS

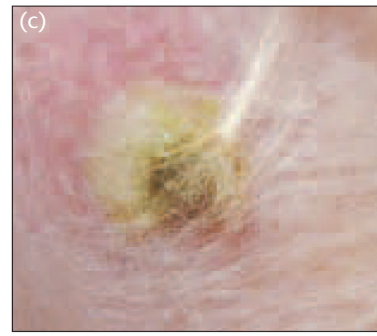
- History of scratching, licking, rubbing, or chewing.
- Clinical signs dominated by secondary pathology: hair shaft damage, lichenification, hyperpigmentation, and excoriation.
- Establish age of onset, initial distribution, seasonality, response to previous treatment including ectoparasite control.
- For an approach to pruritus, see Figure 3.10e.

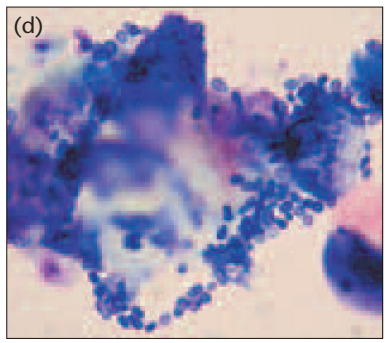
3.10.6 PUSTULES

An intraepidermal vesiculobullous lesion up to 10 mm in diameter filled with pus. May be follicular or interfollicular. The majority are bacterial in origin but some may have an immune-mediated cause and be sterile.

- For an approach to pustules, see Figure 3.10f.







Figures 3.10b,c,d Skin lesions. Interdigital erythemain a dog with atopic dermatitis (Figure 3.10b); non-follicular pustule in a dog with bullous impetigo (Figure 3.10c). Yeast infection. Budding malassezia (oval shaped) in an impression smear from a dog with otitis externa (Figure 3.10d).

3.10.7 ALOPECIA

- There are two basic mechanisms of hair loss: failure of the follicle to produce hair and damage to the hair shaft following emergence.
- Distribution may be diffuse, focal, or multifocal; regional, symmetric, or asymmetric.
- Accompanying clinical signs may include pruritus, self-trauma, erythema, papular, or vesiculobullous eruptions and seborrhoea.
- For an approach to alopecia, see Figure 3.10g.

3.10.8 DISEASES OF THE CLAW AND CLAW FOLD

- Claw disease is usually immune mediated, commonly associated with lupoid onychodystrophy and pemphigus foliaceus. Claw disease has been reported with other causes, including Leishmania and distemper, as well as metastatic lung neoplasia in old cats. Idiopathic disease is described in some dog breeds, e.g. dachshund and Siberian husky.
- Diagnosis is by skin scrapes, hair plucks, radiography, and biopsy.
- Asymmetric claw-fold disease is usually associated with bacterial infection secondary to bite wounds or trauma.
- Neoplasia (squamous cell carcinoma) and dermatophytosis are rare causes.
- Symmetrical claw-fold disorders result from bacterial infection of the claw fold secondary to:
    - Metabolic conditions (HAC, DM, hypothyroidism, necrolytic migratory erythema).
    - Immunosuppression, e.g. FeLV Immune-mediated disease.

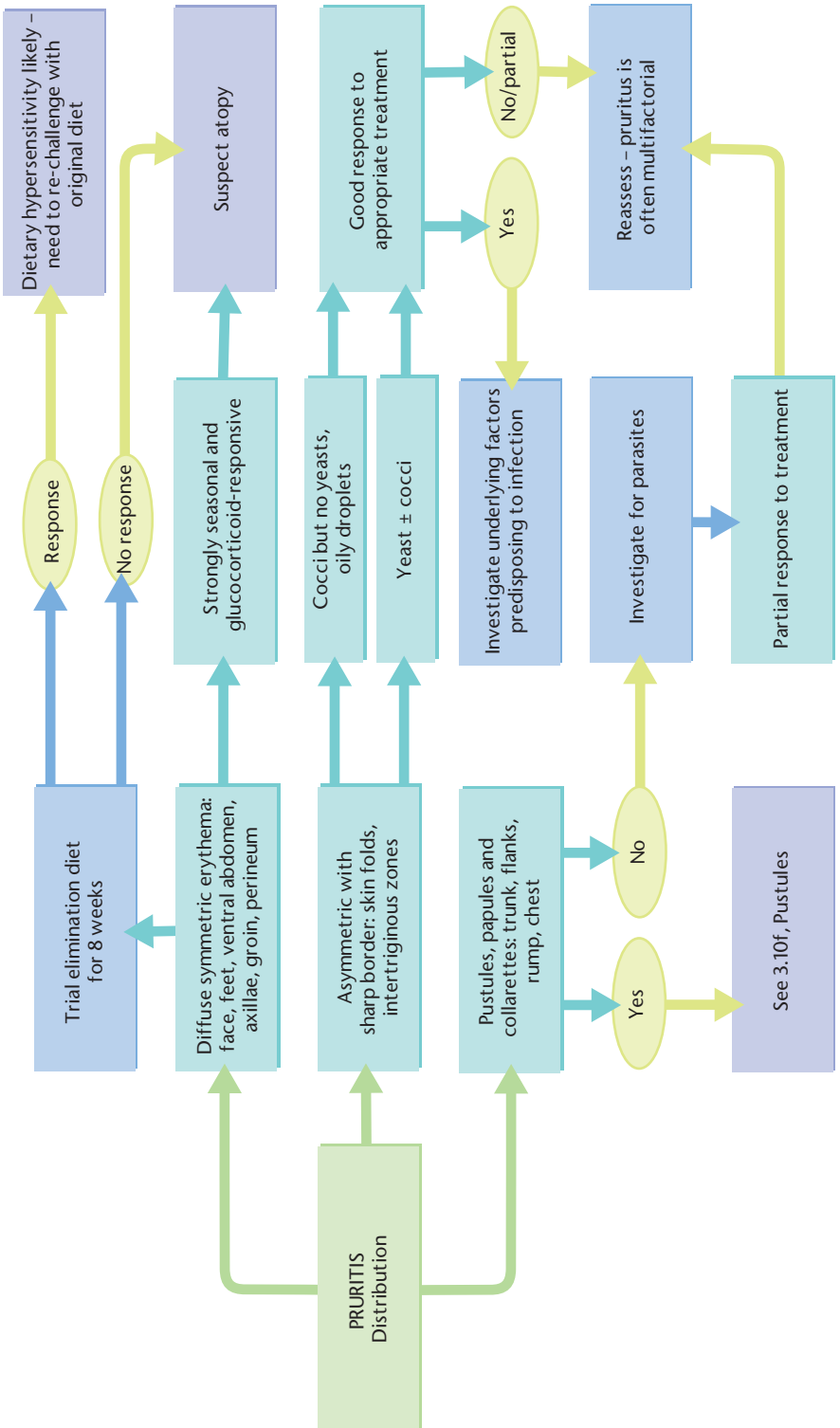

PRURITIS
Distribution
Diffuse symmetric erythema: face, feet, ventral abdomen, axillae, groin, perineum
Asymmetric with sharp border: skin folds, intertriginous zones
Pustules, papules and collarettes: trunk, flanks, rump, chest
Trial elimination diet for 8 weeks
Response
Dietary hypersensitivity likely – need to re-challenge with original diet
No response
Suspect atopy
Strongly seasonal and glucocorticoid-responsive
Cocci but no yeasts, oily droplets
Yeast ± cocci
Investigate underlying factors predisposing to infection
Good response to appropriate treatment
No/partial
Yes
Reassess – pruritus is often multifactorial
No
Investigate for parasites
Partial response to treatment
Yes
See 3.10f, Pustules

Figure 3.10e Algorithm illustrating an approach to the pruritic patient

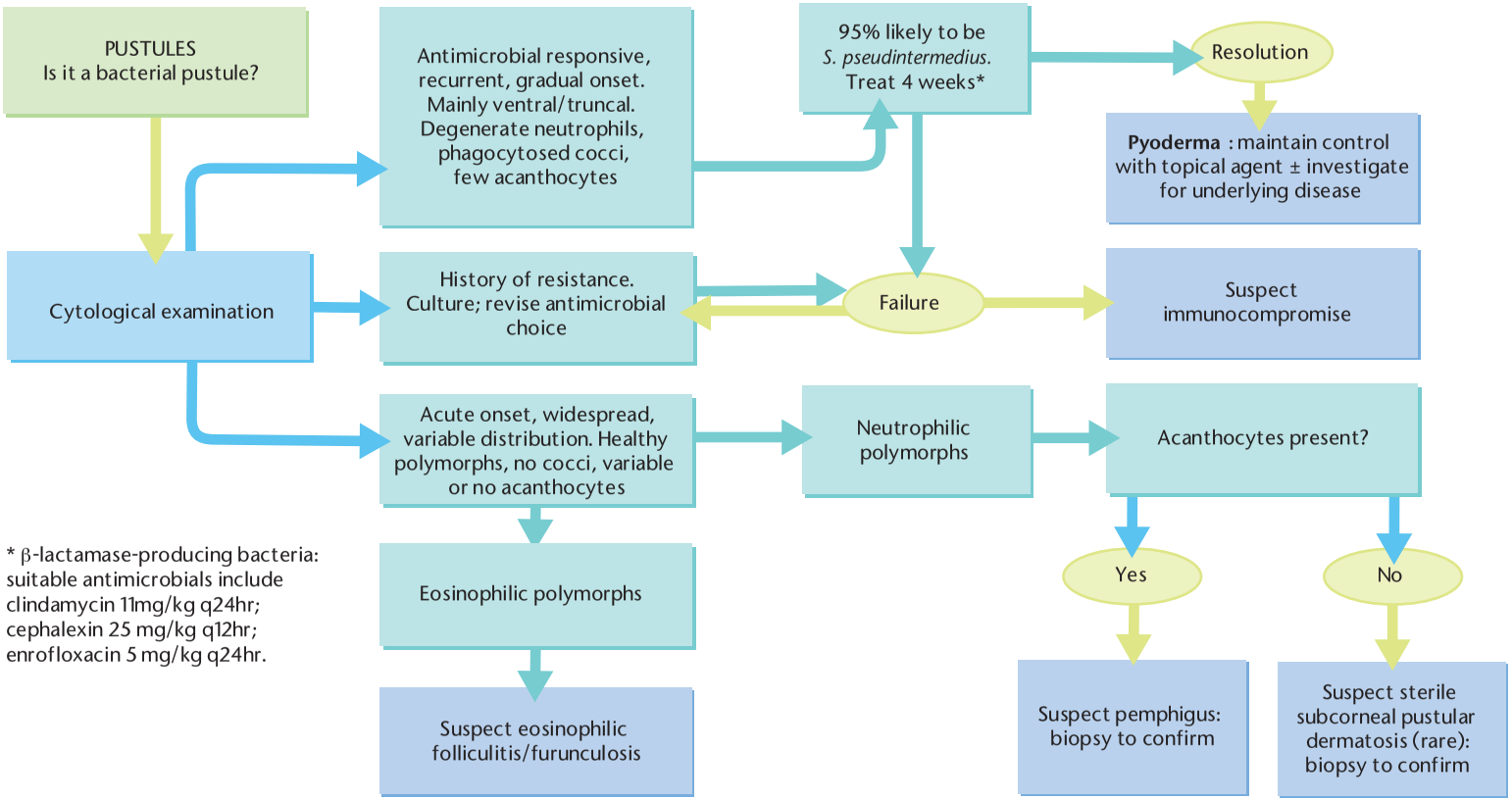

PUSTULES
Is it a bacterial pustule?
Cytological examination
Antimicrobial responsive, recurrent, gradual onset. Mainly ventral/truncal. Degenerate neutrophils, phagocytosed cocci, few acanthocytes
History of resistance. Culture; revise antimicrobial choice
Acute onset, widespread, variable distribution. Healthy polymorphs, no cocci, variable or no acanthocytes
Eosinophilic polymorphs
Suspect eosinophilic folliculitis/furunculosis
95% likely to be S. pseudintermedius. Treat 4 weeks*
Failure
Neutrophilic polymorphs
Resolution
Pyoderma : maintain control with topical agent ± investigate for underlying disease
Suspect immunocompromise
Acanthocytes present?
Yes
No
Suspect pemphigus: biopsy to confirm
Suspect sterile subcorneal pustular dermatosis (rare): biopsy to confirm
* β-lactamase-producing bacteria: suitable antimicrobials include clindamycin 11mg/kg q24hr; cephalexin 25 mg/kg q12hr; enrofloxacin 5 mg/kg q24hr.

Figure 3.10f Algorithm illustrating the approach to a patient with pustules. * a) Start with 4% chlorhexidine shampoos, allowing 10 mins contact 3× week.b) Tier 1 antimicrobials – example cephalexin at 25 mg/kg q12 hr for 4 weeks.c) If this fails, culture is strongly indicated to identify methicillin-resistant *Staphylococcus pseudintermedius* (MRSP).d) Tier 2 antimicrobials if culture results indicate this is appropriate.

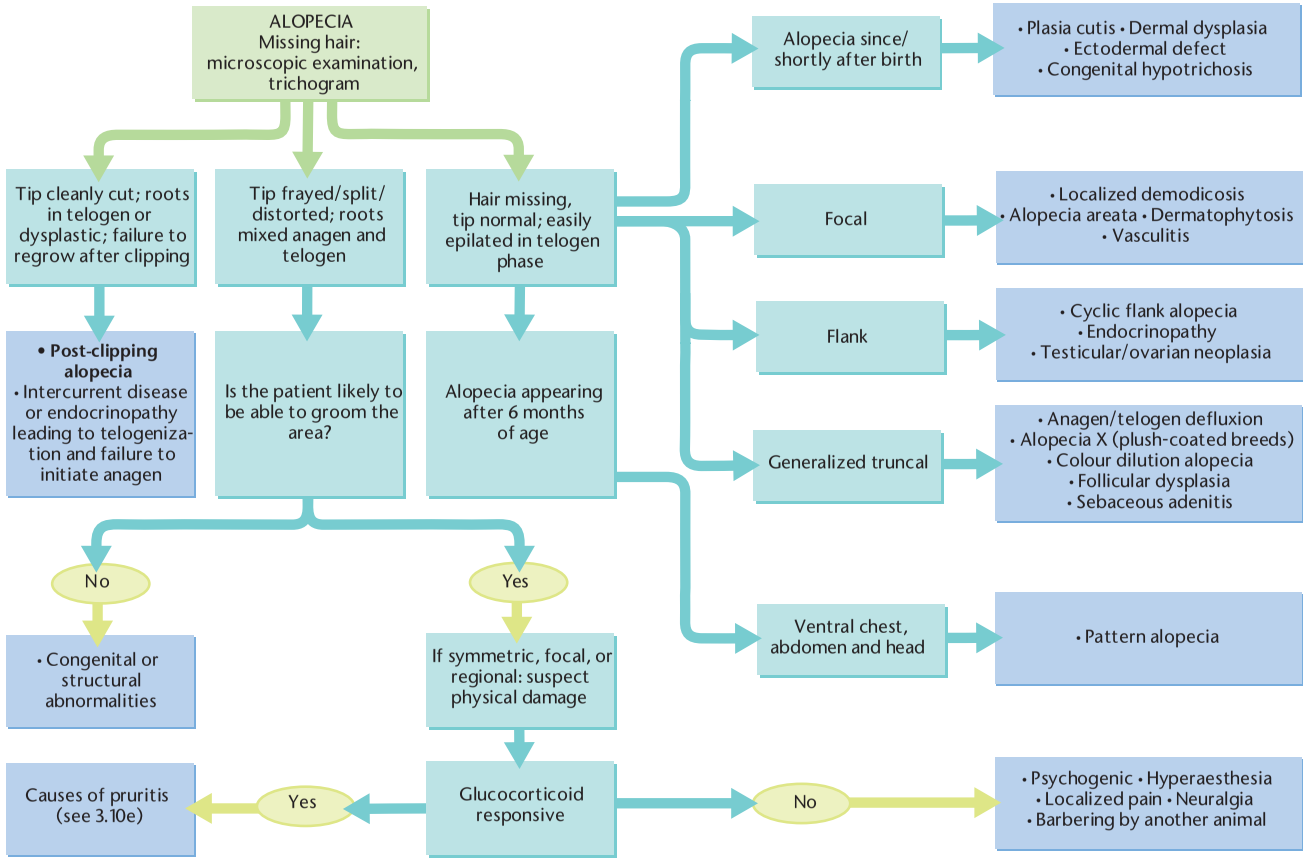

ALOPECIA
Missing hair: microscopic examination, trichogram
Tip cleanly cut; roots in telogen or dysplastic; failure to regrow after clipping
Tip frayed/split/ distorted; roots mixed anagen and telogen
Hair missing, tip normal; easily epilated in telogen phase
• Post-clipping alopecia
• Intercurrent disease or endocrinopathy leading to telogenization and failure to initiate anagen
Is the patient likely to be able to groom the area?
Alopecia appearing after 6 months of age
No
Yes
• Congenital or structural abnormalities
If symmetric, focal, or regional: suspect physical damage
Causes of pruritis (see 3.10e)
Yes
Glucocorticoid responsive
No
• Psychogenic • Hyperaesthesia • Localized pain • Neuralgia • Barbering by another animal
Alopecia since/ shortly after birth
• Plasia cutis • Dermal dysplasia • Ectodermal defect • Congenital hypotrichosis
Focal
• Localized demodicosis • Alopecia areata • Dermatophytosis • Vasculitis
Flank
• Cyclic flank alopecia • Endocrinopathy • Testicular/ovarian neoplasia
Generalized truncal
• Anagen/telogen defluxion • Alopecia X (plush-coated breeds) • Colour dilution alopecia • Follicular dysplasia • Sebaceous adenitis
Ventral chest, abdomen and head
• Pattern alopecia

Figure 3.10g Algorithm illustrating the approach to a patient with alopecia.

TREATMENT

3.10.9 TOPICAL AGENTS

- Topical agents are selected on the basis of detailed, accurate observation of presenting clinical characteristics, supported by cytology.
- Several companies produce similar topical agents and it is best to become familiar with a limited range.
- For ectoparasiticides, see 1.1.7.
- ***General principles***: topical agents must have effective skin contact. This may require long coats to be clipped.
    - Where there is marked scaling or oiliness, this should be removed with keratolytic and keratoplastic washes or degreasing shampoos. This enhances contact, removes surface irritants, antigens, organisms, and the oily environment in which cutaneous pathogens and commensals flourish. Ensure adequate contact time and repeat frequently enough to prevent recurrence.
- ***Dry scaling***: common agents include sulphur, selenium sulphide, salicylic acid.
    - Usually a secondary phenomenon (rarely a primary keratinisation defect).
    - Use combination of keratoplastic/keratolytic washes followed by humectants or emollients.
    - Essential fatty acid supplements or rinses may be useful.
- ***Greasy seborrhoea***: common agents include benzoyl peroxide, coal tar, selenium sulphide.
    - Prewash with degreasing shampoos followed by keratoplastic washes. May have to be repeated.
    - Once the severe problem is controlled, switch to milder agents.
    - Commensal overgrowths are common.
- ***Pyoderma***: common agents include chlorhexidine, benzoyl peroxide, mupirocin (local).
    - Use with systemic treatments or alone for long-term maintenance. In the latter role, benzoyl peroxide can be irritant and drying.
- ***Commensal overgrowth***: common agents include chlorhexidine, benzoyl peroxide, miconazole, ketoconazole.
    - Prewash with degreasing shampoo to remove the oily environment.
    - Guided by cytology, wash with appropriate antimicrobial shampoo.
- ***Pruritus***: common agents include colloidal oatmeal, sodium lactate, coconut oil, essential fatty acids.
    - Relief can be obtained simply by removing organic and inorganic debris, surface antigens, irritants and organisms, and the lipids that bacteria degrade to free fatty acids.
    - Rehydrating the stratum corneum is helpful, as are topicals that replace missing waterproofing components.

NUTRITION

3.10.10 DIETARY APPROACH TO SKIN DISEASE

- Skin and hair reflect the general metabolic health of a patient.
- In crisis, maintenance (especially of hair) is sacrificed for the essential body systems. Good hair and coat quality therefore usually reflects good nutritional status (see Table 3.10a).
- Food-allergic skin disease: cases can be intensely pruritic with secondary damage to the hair/skin. They are managed with exclusion diets as for GIT, but response tends to be slower – at least 6–8 weeks.
- Dietary supplementation with essential fatty acid (EFA): diets should contain at least 3% (dog), 1.5% (cat) EFA on a DM basis. More than one-third of this should be linoleic

Table 3.10a Dietary deficiencies and skin problems	
Deficient factor	**Consequence**
Protein and energy	Keratinization abnormalities; loss of hair colour; secondary bacterial/yeast infection; slow healing; telogen/anagen defluxion
Essential fatty acids	Excessive scaling; alopecia and dry, dull coat; lack of normal growth; erythroderma; interdigital exudation
Zinc	Alopecia; skin/buccal margin ulceration; dermatitis; foot pad and paronychia; slow hair growth; hyperkeratotic plaques; secondary bacterial/yeast infection; slow healing
Copper	Dry/rough coat with loss of colour; reduced density; alopecia
Vitamin A	Seborrhoea; keratinization disorders; hyperkeratosis; cutaneous neoplasia; lamellar ichthyosis; head area particularly affected with acne, and nasal and ear margin change
Vitamin E	Discoid and systemic lupus; pemphigus; vasculitis; acanthosis nigrans; sterile panniculitis; dermatomyositis

acid. Supplementation is 40 mg omega-3/kg/day, aiming for an omega-6: omega-3 ratio in the diet of 5:1–10:1.

- Copper coat: many old dogs will develop copper coats over time. This pigment loss responds to tyrosine supplementation.

3.10.11 OTITIS

- Otitis externa (OE) is a very common presentation in small animal practice. Otitis media is relatively uncommon and otitis interna is usually associated with vestibular signs (see 3.11.3 and Table 3.11b).
- In many cases OE is an extension of a more generalised skin disease meaning that treating the ears alone may not resolve the issue in the long term. The presence of bacteria and yeasts is common in OE but this are usually secondary invaders and whilst treatment will relieve clinical signs recurrence is common and risks, particularly with pseudomonas, ever increasing antimicrobial resistance. In severe disease pathogens can traverse the tympanic membrane resulting in otitis media.
- Common primary causes of chronic OE include:
  - Hypersensitivity.
  - Foreign bodies.
  - Parasitic disease.
  - Keratinisation disorders.
  - Immune-mediated diseases.
- Many dogs with chronic, painful disease will become extremely head shy and reluctant to have their ears examined. Sedation is a better approach than severe manual restraint.
- ***Diagnosis of OE*** typically requires otoscopy and cytological examination of cerumen collected by a cotton bud (Figure 3.10d). Bacterial culture is valuable in selection of appropriate antimicrobial for acute treatment. In some cases, imaging is required, ideally computed tomography.
- Treatment of OE
  - Immediate relief of inflammation, pain, pruritus.
  - Cleaning of the ear canal.
  - Elimination of microbial infection (bacteria and yeasts).
    - For many patients topical treatment is sufficient but for success it is important to discuss with the owner their ability to deliver treatment.
    - Some patients may require systemic glucocorticoids and/or antimicrobials.
  - Managing perpetuating issues.

Surgical salvage

- In some patients with recurrent, severe poorly managed disease medical therapy will fail.
  - Lateral wall resection.
  - Vertical canal ablation.
  - Total ear canal ablation and bulla osteotomy.

Management of chronic OE

- It is important to spot recurrent cases and not treat each episode as an isolated incident. Owners need to understand that ongoing management at some level may be required. The key to successful management are:
  - Try and define and manage the underlying drivers.
  - Most cases are bilateral so both ears should be managed.
  - Improving the condition of the ear canal increasing resilience to future disease.
  - Maintaining a clean ear canal even when ear appears healthy.

3.11 NERVOUS SYSTEM

3.11.1 *Approach to nervous-system disease* 3.11.2 *Performing a screening examination* 3.11.3 *Localisation of lesions within the CNS* 3.11.4 *Diagnostic tests* 3.11.5 *Approach to management of neurologic disease* 3.11.6 *Nutrition and neurologic disease* 3.11.7 *Assessment and management of spinal cord disease* 3.11.8 *Horner's syndrome* 3.11.9 *Muscular disease* 3.11.10 *Neuromuscular disease*

INTRODUCTION

3.11.1 APPROACH TO NERVOUS-SYSTEM DISEASE

- Signs of nervous-system disease are common but can be subtle (early cognitive dysfunction) or difficult to differentiate from behavioural disorders.
- As with all body systems, disease can be associated with primary cause within that system, or secondary to another localised condition e.g. orthopaedic disease or disease elsewhere, e.g. hepatic encephalopathy. See diagnostic algorithms for collapse (5.6) and seizures (5.8).

Decision making

- Is it disease of the nervous system or disease of another body system causing signs consistent with the neurologic presentation? (3.11.2)
- If it is neurologic disease, what is the localisation? (3.11.3 and 3.11.4)
- What disease is causing these neurologic signs? (3.11.4)
- How can I best approach management and treatment? (3.11.5, 3.11.6)
- Finally, 3.11.7 to 3.11.10 deal with selected specific forms of nervous-system disease.

HISTORY AND PHYSICAL EXAMINATION

3.11.2 PERFORMING A SCREENING EXAMINATION

- A checklist for neurological screening should include:
- ***Mental state***: behaviour, loss of learnt behaviour, alertness, and responsiveness.
- ***Cranial nerve (CN) function***: symmetry of function, reflex testing, eyes (Table 3.11a).
- ***Gait*** (walk and trot): lameness, tripping, ataxia.

- ***Postural reactions***: hopping, placing.
- ***Spinal reflexes***: withdrawal (flexor) reflex; patellar reflex; cutaneous trunci reflex; anal reflex. The biceps and triceps reflexes are technically difficult to elicit. Withdrawal reflex should be of equal strength in all limbs. Patellar reflex can alter with age.
- Muscle tone, size and symmetry.

✪ Some cats tolerate neurologic examination poorly.

Deficits identified within 24 hours of seizures may be transient. Any such cases should be re-evaluated later.

✪ Deficits need to be repeatable to be of significance.

3.11.3 LOCALISATION OF LESIONS WITHIN THE CNS

- ***Forebrain lesions*** (cortex, thalamus, and hypothalamus): seizures, behavioural changes, depression/altered mentation, pacing, head pressing, circling.
  - Circling is typical towards the side of the lesion.
  - Postural deficits are typically contralateral to the side of the lesion.
  - Hemiparesis, visual, and menace defects tend to indicate focal or multifocal disease.
- ***Brainstem lesions*** (pons, midbrain, medulla), CN III, V–XII, altered mentation, mild to severe tetraparesis; gait changes are usually more obvious/severe than with forebrain lesions.
  - Gait deficits are ipsilateral to the lesion.
- ***Vestibular signs*** (peripheral – semicircular canals; central –brain stem/cerebellum/thalamus) are head tilt, ataxia, nystagmus and circling (Table 3.11b). CN VII and VIII signs can be present with disease of the bullae.
- ***Cerebellar lesions*** lead to intention tremor, hypermetria, and ataxia with preservation of strength.
- ***Spinal lesions*** can be localised by checking for motor abnormalities and spinal reflexes in each limb separately (see Table 3.11a).
- Differential signs between left and right limbs suggest lateralisation.
- Upper (UMN) and lower (LMN) motor neuron damage result in different deficits (Table 3.11c).
- The presence of the withdrawal reflex relies on the integrity of the local spinal reflex arcs. It does not require ascending sensory pathways to the brain.
- Do not confuse the presence of pain perception (which does require intact ascending spinal pathways to the brain to function) with the presence of a withdrawal reflex.
- Bladder innervation: see 3.8.1.

DIAGNOSIS

3.11.4 DIAGNOSTIC TESTS

- Good physical examination is vital to localising the site of a lesion, directing further diagnostic tests, and interpreting the results.
- Routine haematology and biochemistry (including dynamic bile acids or ammonia) are important to exclude underlying systemic disease.
- ***Imaging***: plain radiographs are generally of limited value except in trauma cases or to rule out non-neurologic causes.
- Advanced imaging is generally necessary to further evaluate the spinal cord (myelography, CT ± myelography, MRI), and brain (MRI, CT in some circumstances).

Table 3.11a Testing the cranial nerves

Cranial nerves		Testing
I	Olfactory (S)	Difficult: smelling a non-irritant substance
II	Optic (S)	Following dropped cotton wool; menace; PLR; negotiating obstacles
III	Oculomotor (MP)	PLR; eye movement and position; induce horizontal nystagmus in both eyes when head moved sideways
IV	Trochlear (M)	Eye movement and position
V	Trigeminal (SM)	Facial and nasal sensation; corneal and palpebral reflex; jaw tone/movement
VI	Abducens (M)	Retraction of globe on corneal reflex
VII	Facial (SMP)	Muscles of facial expression; blink in response to corneal/palpebral reflex; lip retraction in response to pinching; tear production
VIII	Vestibulocochlear (S)	Balance and head tilt; behavioural response to noise; nystagmus
IX	Glossopharyngeal (SMP)	Gag or swallow reflex
X	Vagus (SMP)	Gag or swallow reflex; laryngeal movement
XI	Accessory (M)	Neck muscle mass – difficult to assess
XII	Hypoglossal (M)	Tongue movement and atrophy; resistance to tongue extension

S = sensory; M = motor; P = parasympathetic

Table 3.11b Central vs. peripheral vestibular signs

	Peripheral	Central
Head tilt/ataxia	Yes	Yes
Proprioceptive defects	No	Ipsilateral
Paresis	No	Ipsilateral
Nystagmus	Horizontal or rotary	Horizontal, rotary or vertical

Table 3.11c Effect of spinal lesions

Site of lesion	C1–C5	C6–T2 = brachial plexus	T3–L3	L4–S3 = lumbosacral outflow
Forelimb	UMN	LMN	Normal	Normal
Hind limb	UMN	UMN	UMN	LMN
LMN damage			UMN damage	
• Loss of voluntary motor activity • Loss of reflex motor activity • Loss of resistance to passive movement (tone) resulting in flaccidity • Rapid muscle atrophy • Loss of sensation • Loss of proprioception			• Limited voluntary movement • Intact local reflexes which may be hyperactive (due to loss of higher control) • Increased muscle tone • Loss of sensation • Loss of proprioception	

- Scan design/acquisition is just as important as image analysis, because of the potential for creating artefacts, or for missing pathology.
- ***Other tests***: cerebrospinal fluid evaluation, electromyelography, nerve and muscle biopsies may be required as appropriate.

TREATMENT

3.11.5 APPROACH TO MANAGEMENT OF NEUROLOGIC DISEASE

- Management and therapy are heavily dependent on the location and underlying cause of the neurologic signs.
- Broadly, three options exist:
  - ***Surgery***, to correct anatomic defects or remove structures (disc material, haemorrhage, tumours) that may be causing compression.
  - ***Medical therapy***, most commonly used to manage inflammatory, infectious, and neoplastic disease or seizures and to provide pain relief.
  - ***Physiotherapy***, to maintain/improve function.

NUTRITION

3.11.6 NUTRITION AND NEUROLOGIC DISEASE

- For most neurologic disease, specific nutritional management is not required.
- Nutritional deficiencies causing neurologic signs are rare, e.g. thiamine deficiency, arginine, cobalamin in cats.
- Increasing antioxidant levels (vitamin E omega-3-polyunsaturated fatty acids), mitochondrial cofactors (vitamin B6), and co-enzyme Q, as well as some nutraceuticals (Ginkgo biloba, S-adenosyl methionine phosphatidylserine) may be helpful in managing cognitive dysfunction (CD).
- Medium chain triglycerides (MCT) are also recommended in dogs with canine cognitive dysfunction and in seizure control either as part of the diet or as a supplement.

SELECTED TOPICS

3.11.7 ASSESSMENT AND MANAGEMENT OF SPINAL CORD DISEASE

- The most common presentation relates to difficulty in walking.
- This is usually due to decreased (paresis) or loss (paralysis) of voluntary movement or ability, altered muscle tone (increase or decrease in extensor muscle tone).
- Always consider non-neurological causes of weakness, inability to move, collapse: orthopaedic disease (e.g. hip pain; bilateral cruciate disease; elbow fracture; iliopsoas muscle injury); cardiovascular disease (e.g. anaemia; cardiomyopathy; cardiac arrhythmia; aortic thrombosis); hypotension (sepsis; hypoadrenocorticism; blood loss; intoxication).
- Major differential diagnoses for spinal cord disease are:
  - Often species and breed dependent (signalment), e.g. chondrodystrophic breeds (French bulldog; dachshunds; spaniels) commonly affected by intervertebral disc extrusion, in the cervical and thoracolumbar spinal cord. Malformations are common in brachycephalic breeds, (e.g. subarachnoid diverticulum; caudal facet dysplasia; hemivertebrae) with variable clinical significance.
  - Determined by the localisation of the lesion (examination), e.g. atlantoaxial subluxation present as cervical cord disease, e.g. FIP presents as multifocal disease .

Table 3.11d Spinal cord disease grading

Grading	I	II	III	IV	V
Signs	Spinal pain	Paraparesis	Non-ambulatory paraparesis	Paraplegia; pain perception intact	Paraplegia; pain perception absent

  - Determined by the onset and progression of signs (history), e.g. vascular diseases (e.g. fibrocartilaginous emboli) are sudden in onset and non-progressive, e.g. spinal cord or vertebral neoplasia is typically progressive, but may present acutely, e.g. spinal fractures cause a sudden onset of neurological signs, e.g. degenerative myelopathy is a progressively worsening, non-painful disease.
- ***Physical examination***: see also Table 3.11d and page 131–132.
  - The presence or absence of pain perception in paralysed hindlimbs is a prognostic sign.
  - Pain perception in paralysed limbs signifies the presence of some intact pathways traversing the lesion and hence some remaining cord integrity.
  - Schiff-Sherrington posture (thoracic limb hypertonia/rigidity and paraplegia) occurs with acute, severe thoracolumbar spinal cord lesions and is not a prognostic sign.
  - Do not confuse a cervical cord lesion with a Schiff–Sherrington presentation
  - Cutaneous trunci reflex is useful to map worsening spinal cord damage in myelomalacia.
- ***Diagnosis***
  - The spinal cord lesion must be localised in order to interpret imaging findings.
  - Plain radiographs are helpful in cases of spinal trauma, e.g. RTA.
  - Two views are ideal but not always possible.
  - Myelography is technically challenging and has been largely replaced by MRI.
  - MRI is the best tool for investigating disease of the CNS.
  - CT scan is best for spinal fractures, spinal malformations, surgical planning for internal fixation
- ***Treatment*** for spinal disease is guided by diagnosis, rate of progression of clinical signs, prognosis, and owner's goals and available funds.
  - Options are surgical, conservative (strict cage rest, at home or in hospital depending on severity, continence, etc.) and medical (for spinal trauma) aimed at maintaining CNS perfusion.
  - Management of the paralysed patient:
    - Bladder expression (manual or catheter).
    - Keep clean and dry.
    - Turn frequently.
    - Ensure adequate fluid and calorie intake, as the patient may not be able to reach food/water.
    - Enemas as required.
    - Physiotherapy.
    - Pain relief as required.

3.11.8 HORNER'S SYNDROME

- Sympathetic innervation to the eye is a 3-neuron pathway. Horner's syndrome is classified based upon the level of dysfunction within the oculosympathetic pathway central (first order) , preganglionic (second order) , or postganglionic (third order)
- ***History and physical examination***: miosis of the affected eye is the most commonly identified component of Horner's syndrome and is most pronounced in dim light or darkness.

- Important differentials for miosis include painful conditions of the eye: uveitis; corneal ulcer.
- Other signs may include third eyelid prolapse, enophthalmia, ptosis.
- **Diagnosis**: most cases are third order and idiopathic. A drop of 1% phenylephrine causes pupillary dilation and resolves enophthalmos, third eyelid protrusion, and ptosis in under 20 min, due to denervation hypersensitivity which generally starts after 2–3 weeks.
    - Localisation of the lesion: varies from midbrain through spinal cord to T1–T3 up the vagosympathetic trunk and cranial cervical ganglion next to the tympanic bulla.

3.11.9 MUSCULAR DISEASE

- The majority of myopathies are acquired (Table 3.11e) but some inherited conditions exist, e.g. centronuclear myopathy of Labrador retrievers, or muscular dystrophy in Siamese and Maine coon cats. See Table 3.11e for examples.
- ***History and physical examination***
    - Myopathies are associated with muscle weakness or pain, reduced exercise tolerance, and abnormal (stiff) gait.
    - Sensation is normal: reflexes may be slow due to decreased motor ability.
    - Due to the lack of a nuchal ligament, muscular weakness in cats commonly presents as neck ventroflexion.
- ***Diagnosis*** should be via history, including video of signs, and physical examination.
    - Further tests include muscle enzymes (AST, CPK), electrolytes, triglyceride, cholesterol, retrovirus status, toxoplasma/Neospora titres, 2M and acetyl choline receptor antibodies, muscle biopsy (check handling instructions), edrophonium tests, and electromyography.
- ***Treatment*** for neuromuscular disease is dependent on diagnosis.
- ***Nutrition:*** dietary support with antioxidants and nutraceuticals, e.g. carnitine may be helpful in some diseases.

3.11.10 NEUROMUSCULAR JUNCTION DISEASE

- Function of the neuromuscular transmission can be affected by disease, by drugs such as aminoglycosides, some antimicrobials, e.g. erythromycin, antiarrhythmic agents, phenothiazines, and magnesium, given parenterally or in cathartics and by toxins, e.g. organophosphates, botulism, or snake venom.
- In North America and Australia, tick paralysis is the result of neuromuscular junction dysfunction.
- Myasthenia gravis is the most common junctionopathy due to immune-mediated destruction of acetylcholine receptors (AChR).

Table 3.11e Acquired myopathies

Type	Examples
Infectious	*Toxoplasma*, *Neospora*, *Trichinella*, *Leptospirosis*, *Clostridia*, retrovirus
Immune-mediated	Temporal muscle (2M antibody-positive), polymyositis, dermatomvositis
Endocrine	Hyperthyroid (cat), hypothyroid (dog), hyperadrenocorticism (including iatrogenic)
Metabolic	Hypokalaemia, hyperkalaemia, malignancy, exercise-induced hyperthermia
Neoplastic	Rhabdomy(osarc)oma, paraneoplastic
Other	Following trauma, denervation, or ischaemia; myositis ossificans, fibrotic myopathy, polymyopathy (cat), vitamin E deficiency

- ***Physical examination***: findings may include stiff gait, atrophy of muscles, weakness on exercise (myasthenia) or complete, flaccid paralysis (botulism) – may be generalised or focal.
  - Tendon reflexes are often intact in the early stages of the disease.
  - Can affect muscles of facial, pharyngeal, laryngeal, and oesophageal skeletal muscle.
- ***Diagnosis*** can be difficult; consider serum cholinesterase activity, AChR antibody levels and electromyography.
- ***Treatment*** is symptomatic and supportive: elevated feeding (dysphagia); physiotherapy to prevent muscle contraction antitoxins. Anticholinesterase agents for myasthenia ± immunosuppressive therapy (controversial) ± surgery.

3.12 ENDOCRINE SYSTEM

3.12.1 *Presentation* 3.12.2 *Diabetes mellitus* 3.12.3 *Hypothyroidism in dogs* 3.12.4 *Hyperthyroidism in cats* 3.12.5 *Hyperadrenocorticism* 3.12.6 *Hypoadrenocorticism* 3.12.7 *Differential diagnoses of hyper-and hypocalcaemia* 3.12.8 *Insulinoma* 3.12.9 *Acromegaly*

INTRODUCTION

3.12.1 PRESENTATION

- Endocrine disease is relatively common in cats (hyperthyroidism and diabetes mellitus) and dogs (diabetes mellitus, hypothyroidism, hyperadrenocorticism).
  - Each endocrine disease presents differently and requires different diagnostic testing and treatment.
- The possibility of endocrine disease should be considered in patients with multisystemic signs, such as PU/PD/PP (2.8), weight loss (2.7), inappetence, skin and coat changes (3.10); and especially if disease seems to be affecting a number of organ systems.

3.12.2 DIABETES MELLITUS

- Diabetes is the most common endocrinopathy in cats and dogs. It tends to affect middle-aged dogs and cats.
- Genetic predispositions exist in specific dog and cat breeds.
- Transient diabetes occurs in up to 80% of cats associated with glucose toxicity and can be induced during oestrus in dogs.
- ***Dogs***: nearly always type 1 diabetes due to a failure of pancreatic islet cell production of insulin.
- **Cats**: predominantly type 2, i.e. reduced insulin production due to β-islet cell dysfunction secondary to glucotoxicity with increased end-organ resistance to insulin. If glucotoxicity can be resolved allowing β-islet cell function to improve, remission may be achieved.

History and physical examination

- PU/PD/PP ± weight loss is the most common presentation.
- Less commonly, cases present:
  - Collapsed with vomiting associated with diabetic ketoacidosis.
  - Having gone blind (dogs) due to cataracts.
  - In generally poor condition and with reduced activity and ability to jump (cats).

Diagnosis

- Persistent hyperglycaemia and glucosuria need to be shown.
- Other causes of hyperglycaemia should be considered particularly in cats associated with stress – confirm persistence by elevated fructosamine/HbA1c.
- Glucosuria in the absence of hyperglycaemia can occur in association with renal tubular damage.
- Hyperglycaemia needs to exceed the renal threshold (about 12–15 mmol/l) to cause typical diabetic clinical signs.

Treatment

Insulin

- Virtually all dogs and some cats require insulin therapy to manage their diabetes; biphasic (Caninsulin – UK) or protamine zinc insulin (ProZinc – UK) is an appropriate first choice.
- The starting dose for a newly diagnosed diabetic: 0.25–0.5 unit/kg q12–24 hr.
- The dose should be revised no more frequently than every 5–7 days.
- Longer-acting insulins (protamine or glargine) are needed in some cases, particularly cats.
- Monitor the glycaemic response ideally with flash glucose monitoring system which gives up to 14 days of continuous reading. Value of serial fructosamine or in-clinic glucose curves is questionable. Spot glucose is of virtually no value unless patient clinically unwell, e.g. hypoglycaemic.
- Common causes of poor response to therapy include:
    - Administration problems – syringe filling, insulin quality, injection site.
    - Dose as yet inadequate.
    - Insulin resistance if dose >2 iU/kg: look for intercurrent disease, e.g. urinary infection, pancreatitis, inflammation, neoplasia.
    - Somogyi overswing.

Sodium glucose cotransporter -2 (SGLT-2) inhibitors

- Licensed for use in cats, they act to increase glucose loss in the urine lowering blood glucose and therefore glucose toxicity resulting in improved β-islet cell function and ultimately glycaemic control.
- Single daily oral dosing, standard dose rate based on bodyweight.
- Inhibits SGLT-2 for 2–3 days. Diarrhoea most common side effect, usually mild and self-limiting. Evidence of improvement within 2 weeks in most cats with glucose below the renal threshold.
- Risk of patients developing ketosis and diabetic ketoacidosis (DKA) particularly in the first weeks of treatment, regular (every 2–3 days) monitoring for urine ketones is advised.
- Symptomatic hypoglycaemia has not been reported.
- There are several other adjunctive therapies suggested, such as chromium, acarbose, metformin, sulphonylureas, and herbs. They are rarely indicated and of limited efficacy.

Nutritional management

- ***Dogs***: treat obesity, increase fibre, and digestible carbohydrate. Fat and protein <30% of metabolisable energy; maintain consistent calorie intake; match post prandial glucose rise with
- onset of insulin activity if using biphasic insulin.
- ***Cats***: treat obesity; maintain high-protein/high-fat diet.

Selected topic

Diabetic ketoacidosis

Basic management of a DKA crisis involves:

- Diagnosis – hyperglycaemia, ketosis (blood or urine).
- Acidosis requires in-house capability but can be assumed in a sick patient.
    - Baseline data from blood sample: urea/creatinine, glucose, electrolytes, phosphate, calcium, PCV.
    - Fluid therapy – 0.9% NaCl with 40 mmol/l potassium.
    - Bicarbonate – 0.5 mmol/kg slow IV if HCO3– <12 (dog), <10 mmol/l (cat).
    - Neutral insulin – 0.2 iU/kg IM repeat 0.1–0.2 iU/kg every 1–2 hours.
    - Parameters should be monitored and treatment revised depending on response.

Euglycaemic DKA

- Patients on SGLT-2 inhibitors can develop DKA in the absence of marked glucose elevations. Treatment with insulin is required but it is essential that glucose containing fluid therapy is used.

3.12.3 HYPOTHYROIDISM IN DOGS

Introduction

- Hypothyroidism can be congenital (rare) or acquired, secondary to immune-mediated thyroiditis.

History and physical examination

- The onset of clinical signs can be subtle and slow (Table 3.12a)

Diagnosis and testing

- Diagnosis is based on appropriate history, supporting clinical signs, biochemical changes (mild non-regenerative anaemia, raised cholesterol and triglycerides, elevated ALKP), consistent
- thyroid function tests, and response to therapy.
- Total T4 is lowered by non-thyroidal illness (NTI), drugs (corticosteroids, sulphonamides, barbiturates), breed (e.g. sight hounds), age, and time of day.
- Free T4 is less affected by non-thyroidal factors but needs to be measured by equilibrium dialysis.
- TT4 results (Table 3.12b) alone should not be used alone to make a diagnosis of hypothyroidism:
    - Low TT4 and high *c*TSH is consistent with hypothyroidism.
    - 20–40% of dogs with low TT4 and normal *c*TSH are hypothyroid.
    - High *c*TSH with normal TT4 is associated with early hypothyroidism, recovery from NTI, or use of certain drugs.
- Dynamic testing is rarely necessary, but a TSH stimulation test is the most useful.
- Autoantibodies:
    - Thyroglobulin autoantibodies are a marker for subclinical lymphocytic thyroiditis and future hypothyroidism.
    - T3 and T4 autoantibodies give spuriously elevated radioimmunoassay results in dogs that may be hypothyroid.

Table 3.12a Clinical signs of hypothyroidism in dogs

Metabolic	Lethargy; dullness; exercise intolerance; obesity; hypothermia
Skin	Seborrhoea; alopecia; dorsal scaling; coat is dull, bleached, brittle; refractory pyoderma; hyperpigmentation; comedones
Neuromuscular	Peripheral polyneuropathy; peripheral vestibular signs; ataxia; seizures; coma; myxoedema; myopathy
Rare	Cardiovascular; reproductive; ophthalmic; behavioural; skeletal (cretinism)

Table 3.12b Interpretation of TT_4 results

Serum total T_4 concentration (µmol/l)	Likelihood of hypothyroidism
>26	Very unlikely
20–26	Unlikely
14–20	Equivocal
7–14	Possible
<7	Likely

Treatment

- Treatment is by l-Thyroxine (levothyroxine) hormone replacement, via liquid and tablet preparations.
  - The starting dose is 20 µg/kg q24 hr or 10 µg/kg q12 hr.
- Monitor response – T4 and cTSH level 6 hours post medication ideally shows T4 in the upper half of the reference interval and a low TSH. If not, alter dose as necessary. Full response can take 6–8 weeks. If there is no response at this stage, re-evaluate the diagnosis.
- ***l-Thyroxine trial*** – can be difficult to interpret, many dogs will show improved activity and some hair growth regardless of thyroid status.

3.12.4 HYPERTHYROIDISM IN CATS

Introduction

- Hyperthyroidism is a very common endocrinopathy in older cats, but there is marked regional variation.

History and physical examination

- Presenting clinical signs include weight loss (90% of cases); polyphagia (60%); PU/PD (50%); hyperactivity (40%); GIT signs (40%); poor coat quality (35%); respiratory signs (25%); inappetence (15%); lethargy (10%); weakness (10%).
- Rare signs (<10% of cats) include tremors/seizures, heat intolerance, haematuria, and neck ventroflexion.
- On physical examination, >90% have one or more of: a palpable goitre, tachycardia, gallop rhythm, skin changes, hyperthermia, dehydration/cachexia.

Diagnosis and testing

- See Figure 3.12.
- Common clinical pathology changes include elevated red cells with macrocytosis, leucocytosis–neutrophilia, eosinophilia; increased ALT, AST, ALKP, LDH, phosphate and glucose; and azotaemia.

Treatment

- The choice of treatment will depend not only on individual patient factors, but also on the owner's finances and philosophy.
  - ***Radioactive iodine***: generally curative with low side-effect rates, but extended hospitalisation required; moderate risk of hypothyroidism.
  - ***Surgery***: generally curative with moderate risks and recurrence rates.
  - ***Anti-thyroid drugs***: moderate side-effects; needs to be continued indefinitely.
  - ***Diet management***: consider for specific cases.

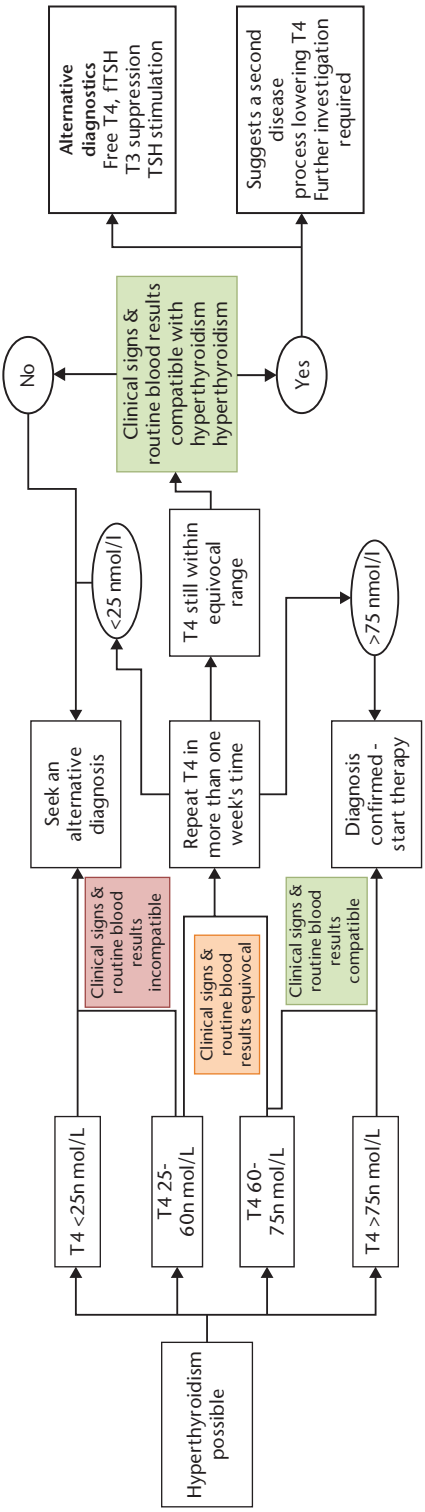


Figure 3.12 Algorithm for the diagnosis of hyperthyroidism in cats.

3.12.5 HYPERADRENOCORTISM (CUSHING'S DISEASE)

Introduction

- Hyperadrenocortism (HAC) is uncommon in cats, but occurs in pituitary and adrenal forms in dogs, with a female bias; breed predispositions vary with region – UK: Bichon frise, Border terrier and miniature schnauzer.

! If clinical signs/pathology are poorly supportive, but adrenal function tests are abnormal, it is easy to make a false diagnosis of hyperadrenocorticism.

History and physical examination

- Clinical signs include PU/PD/PP, abdominal enlargement, exercise intolerance, panting, lethargy, obesity, alopecia, calcinosis cutis, anoestrus/testicular atrophy, comedones, and thin skin.

Diagnosis and testing

- Clinical pathology includes stress leukogram, raised ALKP (can be marked), ALT, cholesterol, bile acids, glucose, low urine SG often with proteinuria and UTI. 25% of HAC dogs hypertensive.
- Adrenal function testing is unreliable: sensitivity and specificity are around 80–90%. There are no definitive tests and all can have false positive and negative results.
    - Consider urine creatinine:cortisol ratio (for a negative rule-out), ACTH stimulation or low-dose dexamethasone suppression test (LDDST) (see Table 3.12c for interpretation of LDDST).
    - Distinguish between pituitary-dependent and adrenal-dependent forms of HAC with endogenous ACTH level, or imaging.

- ***Imaging***: radiographs may show hepatomegaly, good abdominal contrast, potbelly, and adrenal calcification.
    - Measurement of adrenal glands on ultrasound: if left >7.4 mm and/or right >8.1 mm), this is 70–80% specific and sensitive for hyperadrenocorticism.
    - Abdominal CT for large dogs and to assess whether adrenomegaly or adrenal mass and if invasive. CT or MRI can be used to show adenoma of the pituitary.

Treatment

- Initial management: PDH therapy via trilostane (starting dose 2–4 mg/kg q24 hr) or mitotane manages the clinical signs but does not treat the disease.
- For ADH, perform surgery, if possible, if not then treat as PDH; perioperative mortality is high (≈ 25%).

Table 3.12c Interpretation of LDDST

	Result at 3 or 4 hours			
Result at 8 hours	<40 nmol/L & < 50% baseline	<40 nmol/L & >50% baseline	>40 nmol/L & >50% baseline	>40 nmol/L & <50% baseline
Cortisol <40 nmol/L & < 50% baseline	No evidence to support HAC	HAC unlikely	Inverse pattern, HAC unlikely	
Cortisol <40 nmol/L & > 50% baseline	HAC unlikely – how compatible are signs?		Equivocal result; HAC possible	
Cortisol >40 nmol/L & > 50% baseline	PDHAC likely	Consistent with HAC		
Cortisol >40 nmol/L & <50% baseline – interpret in conjunction with clinical signs	PDHAC likely		HAC likely	

3.12.6 HYPOADRENOCORTISM (ADDISON'S DISEASE)

Introduction

- This is an immune-mediated disease, most common in middle aged female dogs (poodles, West Highland white terrier, Great Dane, bearded collie, basset hound are predisposed). It is a rare disease in cats.

History and physical examination

- Clinical signs tend to be non-specific and to wax and wane.
- They include inappetence, lethargy/depression and weight loss.
- Other common signs include GIT signs, shaking, shivering, PU.
- Rarely present collapsed with Addisonian crisis.

Diagnosis and testing

- Clinical pathology is likely to show several of: lack of a stress leukogram, low sodium and elevated potassium, azotaemia, hypoglycaemia, non-ionised hypercalcaemia, low urine SG.
- The definitive test is ACTH stimulation of cortisol and aldosterone.

Treatment

- Initial management is by:
  - Fludrocortisone (0.01 mg/kg PO q12 hr).
  - Desoxycorticosterone pivalate (2 mg/kg IM or SC monthly) plus prednisolone (0.25–0.5 mg/kg PO q24 hr).
- Crisis management is by fluid therapy (0.9% NaCl).

Nutritional management

- Rarely, salt supplementation to the diet is required to maintain sodium levels – initially 0.5 mg/kg/day.

! Do not use 'low salt' type products, as these contain potassium chloride – fatalities have been reported.

3.12.7 DIFFERENTIAL DIAGNOSES OF HYPER- AND HYPOCALCAEMIA

- ***Hypercalcaemia***
  - Parathyroid disease: primary, secondary CKD.
  - Toxic (hypervitaminosis D) – due to excess supplements, ingestion of vitamin D-based creams, calciferol rodenticide.
  - Neoplastic: lymphoma, anal sac adenocarcinoma, myeloma, bone metastasis.
  - Non-ionised hypercalcaemia seen with hyperalbuminaemia, haemoconcentration, hypoadrenocorticism.
  - Granulomatous and fungal disease.
  - Acidosis.

Presentation: most commonly PU/PD, inappetence and lethargy.
Assessment measurement of ionised calcium, PTH, PTHrP, and vitamin D in conjunction with history, physical, and routine laboratory results.

- ***Hypocalcaemia***
  - Hypoalbuminaemia, hypomagnesaemia, spurious due EDTA/oxalate contamination.
  - Renal secondary hyperparathyroidism.
  - Hypoparathyroidism, parathyroidectomy.
  - Diet – inadequate vitamin D, excess phosphorus (all lean meat).
  - Excess phosphate – IV administration, phosphate enema.
  - Malabsorption, pancreatitis.
  - Ethylene glycol poisoning.
  - Eclampsia, alkalosis, C-cell thyroid tumours.

Acute management is required if the patient is agitated/hyperaesthetic use calcium gluconate (10%) 0.5–1.5 (dog), 1–1.5 (cat) ml/kg slow IV over 20–30 minutes, then monitor.
Chronic management with oral calcium 15–25 mg/kg elemental calcium q6–8 hr with vitamin D (calcitriol or alphacalcidol).

3.12.8 INSULINOMA

- Insulinoma is the most common pancreatic tumour in dogs but is rare in cats.
- Cases present with weakness/exercise intolerance, sometimes collapsed or seizuring.
- ***Diagnosis*** is based on a raised insulin/glucose ratio and ultrasound/CT of the pancreas.
    - Other causes of hypoglycaemia should be considered, e.g. liver failure, septicaemia, delayed measurement.
- ***Treatment***: initial stabilisation is with glucocorticoids, and frequent small meals containing complex carbohydrate. After this, surgery carries the best prognosis, especially if there is no gross metastasis.

3.12.9 ACROMEGALY

- Acromegaly is thought to be the cause of insulin resistance in ≈10% of diabetic cats. Occurs uncommonly in non-diabetic cats.
- ***Clinical signs*** various and can appear normal body shape and size
    - Weight gain despite poorly controlled DM.
    - Change in body shape – increased size, enlarged abdomen and head, prognathia.
    - Arthropathies.
    - Organomegaly including cardiac hypertrophy, liver, kidney, spleen, and tongue – stridor and upper airway obstruction.
- ***Diagnosis*** insulin-like growth factor (IGF)-1 elevated >1000 ng/ml.
- ***Treatment*** hypophysectomy, irradiation, somatostatin analogues.

3.13 OBESITY

3.13.1 *Definition and prevalence of obesity* 3.13.2 *Disease risks associated with obesity* 3.13.3 *Body condition scoring* 3.13.4 *Testing for medical causes of obesity* 3.13.5 *Exercise* 3.13.6 *Drug management of obesity* 3.13.7 *Modifying pet and owner behaviour* 3.13.8 *Weight-loss targets*

INTRODUCTION

3.13.1 DEFINITION AND PREVALENCE OF OBESITY

- Pets are defined as overweight when they are 10–20% above their optimal weight.
- 15–20% of cats are estimated to be overweight, and 15–30% of dogs.
- Pets are defined as obese when they are more than 20% above their optimal weight.
- 10% of cats and 10–15% of dogs are estimated to be obese.
- Recommended weight ranges for various breeds exist, but the range can be wide, especially within breeds that have significant size variations, e.g. between working and show Labradors.

HISTORY AND PHYSICAL EXAMINATION

3.13.2 DISEASE RISKS ASSOCIATED WITH OBESITY

- History and physical examination should focus on evidence of systemic disease. Disease risks associated with obesity include:
    - Degenerative joint disease.
    - Respiratory difficulties.

- Decreased cardiac reserve.
- Insulin resistance.
- Poorer response to infectious disease.
- Fatty infiltration of the liver.
- Increased surgical/anaesthetic risk.
- Feline lower urinary tract disease.

3.13.3 BODY CONDITION SCORING

- It is relatively easy to teach owners to evaluate their pets; their score can be compared with yours at each consultation.
- WSAVA recommends a nine-point scoring system (Figure 3.13), with assessment of a four-point muscle condition score – normal muscle mass, mild, moderate, and severe muscle loss https://wsava.org/wp-content/uploads/2020/01/Muscle-Condition-Score-Chart-for-Dogs.pdf

DIAGNOSIS

3.13.4 TESTING FOR MEDICAL CAUSES OF OBESITY

- While physical examination is generally all that is required, if other intercurrent disease is suspected, routine haematology and biochemistry may be appropriate.
- Active management of cases with a body condition score (BCS) ≥7/9 is necessary. In a limited number of cases, specific endocrine testing may be appropriate for:
  - Hypoadrenocorticism (3.12.6)
  - Acromegaly (3.12.9)
  - Hypothalamic disorders
  - Insulinoma (3.12.8)
  - Hypothyroidism (3.12.3)
  - Hypopituitarism.

TREATMENT

3.13.5 EXERCISE

- Exercise helps to change the balance between calorie intake and expenditure.
- A patient's ability to exercise may be affected by their obesity and concurrent disease, such as degenerative joint disease – consider non-weight-bearing exercise, e.g. swimming.
- Optimum regimes for weight loss have not been defined, but limited studies suggest that even short periods (5–10 minutes) of exercise twice daily can have a significant impact.

3.13.6 DRUG MANAGEMENT OF OBESITY

- Medical treatments for obesity have been licensed for use in dogs in the UK but did not gain traction and are no longer available. Dirlotapide (Selentrol) is available in some countries.

NUTRITION

3.13.7 MODIFYING PET AND OWNER BEHAVIOUR

- Most owners know their pet is obese, but don't know how to deal with it effectively. A combination of options should be considered, addressing the causes of overfeeding in particular:
  - Lack of adjustment to individual needs.
  - Energy intake from snack/treats ignored.

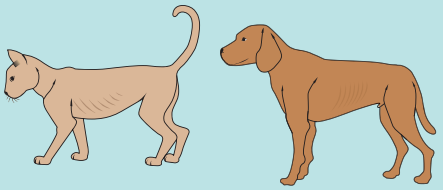
BODY CONDITION SCORING

1: Emaciated

Ribs, spine, pelvic bones easily visible/palpable

Obvious loss of muscle mass and no palpable fat over ribcage

Severe abdominal tuck

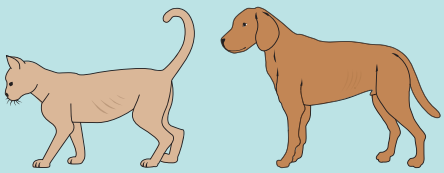


3: Thin

Ribs, spine, pelvic bones visible/easily palpable

Obvious waist

Minimal abdominal/ ribcage fat

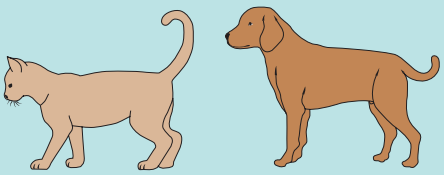


5: Ideal

Ribs, spine, pelvic bones not visible but easily palpable

Obvious waist

Little abdominal/ ribcage fat

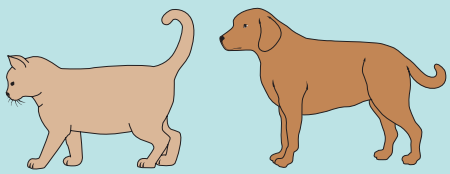


7: Overweight

Ribs, spine, pelvic bones not easily palpable

Loss of waist

Obvious abdominal distension and ribcage fat



9: Obese

Marked thoracic, spinal and abdominal fat deposits

Marked abdominal enlargement

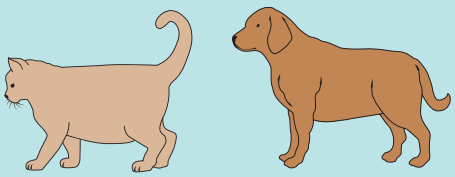


Figure 3.13 Body condition scoring.

  - Eating encouraged as 'a sign of good health'.
  - Begging behaviour indulged.
  - Food provided when the pet is left alone.
  - Excessive use of food as a training aid.
  - Association with owner's attempts to diet.
- Weight-loss diets are available that are nutritionally balanced, with low calorie density, to increase satiety when fed. These diets are easier for most owners than a reduction in the patient's normal ration and do not risk micronutrient deficiency.

✪ Owner support and regular follow-up increase success rate.

3.13.8 WEIGHT-LOSS TARGETS

- Safe weight loss is 1–1.5% per week, to achieve a 15% loss in 15–18 weeks.
- An initial target loss of 15% should be set, with calorie intake at 60% of the maintenance energy requirement (MER) for the target weight (see 1.1.8 for calculation).
- If weight loss is not occurring, consider lack of household compliance; alternative food sources; endocrinopathy; insufficient time allowed. If these can be ruled out, then recalculate and decrease calorie intake by a further 15%.
- Weight clinics are an important part of achieving success as they help support and motivate carers.

3.14 INFECTIOUS DISEASE

3.14.1 *Scope of this section* 3.14.2 *Feline infectious disease* 3.14.3 *Canine infectious disease* 3.14.4 *Overview of antimicrobial treatment* 3.14.5 *Drug choice by body system* 3.14.6 *Oral absorption of antimicrobials* 3.14.7 *Patient considerations* 3.14.8 *Side-effects and toxicity of antimicrobial groups* 3.14.9 *Potential toxicity by body system* 3.14.10 *Vector-borne disease*

INTRODUCTION

3.14.1 SCOPE OF THIS SECTION

- This section focuses on common infectious diseases of dogs and cats. It is clearly not possible to cover all infectious diseases (see Further Reading, page ***). References to certain other infections appear under each body system as appropriate.

COMMON DISEASES

3.14.2 FELINE INFECTIOUS DISEASE

- Viral infectious disease is relatively common in cats (Table 3.14a), especially in kittens and cats held in large groups.
- Carrier states exist for a number of these infections with reactivation in periods of stress or reduced immune function.

3.14.3 CANINE INFECTIOUS DISEASE

- Viral infections are comparatively rare in dogs in the UK, due to vaccination, but outbreaks do occur in unvaccinated groups (Table 3.14b).

Table 3.14a Common Feline Infectious Diseases

Disease	Leukaemia virus	Viral respiratory infection	Immunodeficiency virus	Infectious peritonitis virus
Organism	Oncogenic retrovirus	Herpes or calcivirus	Lentivirus	Coronavirus
Route of infection	Close contact, saliva; in utero; nursing; contaminated blood	Oronasal, conjunctival	Saliva (biting, fighting); perinatal	Oro-nasal; saliva; litter tray; in utero
Incubation	2–4 wks: non-specific signs; 2 yrs: FeLV-related disease	2–5 days	3–6 wks: non-specific signs; 2–5 yrs: FIV-related disease	Experimentally:5–10 days;Field: very variable;stress as a trigger
Infective period	All the time the cat is viraemic: poor survival in the environment	During viraemic carrier state	All the time cat is viraemic; poor survival in the environment	Sporadic occurrence in cat colonies, rarely epidemic
Clinical signs	Lymphoma,leukaemia (rare); immunosuppression and PUO; auto-immune disease; bone-marrow suppression, especially anaemia; uveitis; gastroenteritis	Pyrexia,ptyalism,sneezing,oculonasal discharge, mouth ulceration,keratitis	Chronic infection; ocular disease; stomatitis; wasting and diarrhoea;skin infection; renal failure	Can mimic almost any disease; cavity effusions (wet); granulomatous inflammatory disease (dry); uveitis; CNS signs; fever; renal; hepatic (jaundice); GIT
Diagnosis	FeLV antigen ELISA; VI; PCR; immunofluorescence	Culture or PCR	FIV antibody; ELISA; PCR	No definitive test available Based on haematology and biochemistry results, coronavirus titre, aspirates, fluid or tissue sample
Treatment	Supportive	Supportive	Supportive; maintenance of general health	GS441524 nucleoside analogue oral and injectable giving ~80% survival rate
Control	Test and removal; vaccination; avoid exposure, even of vaccinated cats	Vaccination; environmental controls, e.g. sneeze barriers	Isolation of carriers; avoid saliva/blood transfer, e.g. via feeding bowls	Perinatal hygiene; avoid multiple stressors in kittens; review breeding lines with high incidence of FIP
Prognosis	Poor once clinical signs are apparent	Generally good	Slow, chronic disease course	Very poor once clinical signs are apparent
Vaccine	Moderately successful	Moderately successful	USA: efficacy questionable	Europe: efficacy questionable

Table 3.14b Common Canine Infectious Diseases

Disease	Parvovirus	Distemper	Leptospirosis
Organism	Canine parvovirus 2	Morbillivirus	Spirochete bacteria
Route of infection	Faecal–oral route; direct or indirect contact	Aerosol or droplet exposure	Ingestion; bite wounds; contaminated food or water
Incubation	5–6 days	3–10 days; 2nd fever spike 2 wks	5–7 days
Infective period	3–6 days after recovery; viable virus 3–6 months	7 days post-infection until 1–2 wks (max. 90 days) post-recovery	Months–years following recovery. Killed by drying
Clinical signs	Dull; anorexic; vomiting; haemorrhagic diarrhoea; puppies infected <6 wks old – myocarditis and sudden death	Dull, anorexic; diarrhoea; cough and catarrhal oculonasal discharge; 50%develop neurologic signs	Peracute:death, shock, DIC Subacute:jaundice, DIC Chronic: progressive renal or hepatic disease
Diagnosis	Panleucopenia; serology; virus isolation; faeces; histopathology	Leucopenia; inclusion bodies (conjunctiva); serology; virus isolation	Urine: dark-field microscopy or immunofluorescence Serology
Treatment	Symptomatic and supportive: fluid; nutrition; plasma; blood; antimicrobials;antiemetics; gastric protectants	Symptomatic and supportive: fluid; nutrition; antimicrobials; gastric protectants; antiemetics; antiseizure	Supportive: ensure adequate urine output; gastric protectants. Penicillin acutely; tetracyclines to prevent carrier
Control	Highly infectious: cases need to be isolated	Highly infectious, but poor environmental survival of virus	Control of reservoir hosts
Prognosis	Good if aggressive early care	Guarded	Guarded
Vaccine	Highly effective	Highly effective	Moderate: only some serovars

ANTIMICROBIAL TREATMENT

3.14.4 OVERVIEW OF ANTIMICROBIAL TREATMENT

- The type (especially off-licensed human antimicrobials), duration of treatment and frequency of use should be limited wherever practical to prevent antimicrobial resistance (AMR) developing. Practice policy should be developed various resources available such as Protect Me.
- The main cause of apparent antimicrobial failure is that bacterial infection is not the primary pathology.
- Penetration to 'protected' sites requires movement across epithelial/mesothelial layers and is dependent on:
  - Lipid solubility: if highly lipid soluble, e.g. doxycycline, antimicrobial can easily cross membranes (Table 3.14c).
  - Protein binding: affects drug availability to cross membranes, e.g. beta-lactams.

Table 3.14c Lipophilicity of Antimicrobial Drugs

Lipophilicity	Drug
Acidic low lipophilicity drugs	Penicillins, cephalosporins
Basic, low lipophilicity	Aminoglycosides
Weak acid, moderate lipophilicity	Sulphonamides
Weak base, moderate lipophilicity	Lincosamides; erythromycin
Amphoteric, moderate lipophilicity	Tetracyclines
Highly lipophilic	Fluoroquinolones; doxycycline; chloramphenicol; metronidazole

- The 'tightness' of the barrier itself: the blood–CSF barrier is very tight, so only small molecules, lipid-soluble antimicrobials such as sulphonamides or fluoroquinolones will penetrate into the CSF.
- Acid/base conditions of the site: basic antimicrobials, e.g. lincosamides, macrolides, are ion-trapped in the alkaline environment of the prostate.

3.14.5 DRUG CHOICE BY BODY SYSTEM

- A variety of alternatives are usually available for each body system: choice between them is based on the likely bacterium involved.
  - Bone: amoxyclav, cephalosporin, clindamycin, lincomycin.
  - Urinary tract: potentiated sulphonamides, penicillins, cephalosporin, tetracycline.
  - Prostate: clindamycin, fluoroquinolone, doxycycline, trimethoprim.
  - Hepatobiliary: aminoglycosides, penicillins, cephalosporins, clindamycin, fluoroquinolone, metronidazole.
  - Respiratory: lipid-soluble drugs recommended.
  - CNS: chloramphenicol, potentiated sulphonamides, fluoroquinolone, metronidazole.
- 'Four quadrant cover': for critical cases where there may be infection with a wide variety of bacteria that are both Gram positive and negative, aerobes and anaerobes, e.g. septic peritonitis. This consists of a combination of:
  - Amoxyclav or cephalosporins (group I or II) or gentamicin.
  - Fluoroquinolone.
  - Metronidazole or group IV cephalosporin (cefoxitin).

3.14.6 ORAL ABSORPTION OF ANTIMICROBIALS

- Poor or variable absorption may be noted for the following:
  - Ampicillin: decreased by feeding.
  - Erythromycin: may be a gastric irritant.
  - Tetracyclines: inhibited by milk.
  - Third generation cephalosporins.
  - Aminoglycosides.

3.14.7 PATIENT CONSIDERATIONS

- For neonates, increase initial dose and lengthen the interval.
- The presence of other disease may affect the appropriate antimicrobial, dose or frequency (Table 3.14d).

Table 3.14d Antimicrobial use in renal and hepatic disease

	Renal disease	Hepatic disease
Not affected	Doxycycline; clindamycin; erythromycin	
Decrease dose	Aminoglycosides; sulphonamides; quinolones; cephalosporins	Cephalosporins; metronidazole; doxycycline
Double interval	Penicillins; lincomycin	
Contraindicated	Neomycin; tetracyclines; polymyxin; nitrofurantoin	Chloramphenicol; clindamycin; tetracyclines

3.14.8 SIDE-EFFECTS AND TOXICITY OF ANTIMICROBIAL GROUPS

- Side effects are surprisingly common in patients given antimicrobials with around 40% of dogs showing GI signs (inappetence, vomiting, diarrhoea).
- Aminoglycosides: nephrotoxic, ototoxic, affect neuromuscular junction, e.g. GIT motility. Take care with streptomycin in cats.
- Tetracyclines: catabolic; may cause GIT upset, especially in cats; tooth discoloration; hepatotoxic – take care if patient is azotaemic; oesophageal stricture in cats.
- Quinolones: GIT upset; arthropathy; blindness; seizures; retinal degeneration in cats.
- Sulphonamides: keratoconjunctivitis sicca (KCS); polyarthropathies; skin eruptions; thrombocytopenia; Dobermans especially at risk.
- Erythromycin: may cause vomiting, so give with food, but note that this decreases absorption.
- Metronidaxzole – vomiting, CNS toxicity, hepatotoxicity, neutropenia, haematuria
- Furantoins: GIT upset; hepatotoxic.
- Chloramphenicol: anaemia; dose-dependent; non-regenerative anaemia, especially in cats.

3.14.9 POTENTIAL TOXICITY BY BODY SYSTEM

- Nephrotoxicity:
  - Proximal tubule – aminoglycosides, polymyxins, tetracyclines.
  - Crystalluria – sulphonamides.
- Hepatotoxicity:
  - Parenchymal damage – tetracyclines; erythromycin.; metronidazole
- Neurotoxicity:
  - Ototoxic – aminoglycosides; sulphonamides; lincosamides.
- Neuromuscular blockade: aminoglycosides.
- Blindness in cats: fluoroquinolones.
- Seizures (in susceptible patients): fluoroquinolones, metronidazole.
- Cardiovascular: aminoglycosides; chloramphenicol; lincosamides; sulphonamides.
  - Tetracyclines cause transient depression of output and vasodilation, so avoid rapid IV injection.
- Gastrointestinal tract:
  - Mucosal damage – ampicillin; lincomycin; clindamycin.
  - Vomiting – erythromycin.
  - Oesophageal stricture – doxycycline; clindamycin.
- Bone marrow toxicity: chloramphenicol.
- Arthropathy: fluoroquinolones.

3.14.10 VECTOR-BORNE DISEASE

- Climate change is having a significant impact on the geographic distribution of a number of vector-borne diseases. Increases temperatures allowing vector range to increase and infectious maturation to occur.

- For most vector-borne diseases both antigen/PCR and antibody tests exist. Although more sensitive and specific, antigen/PCR tests are only positive if there is active or recent infection in the tissue sampled. Antibody tests give evidence that the patient has been infected but current status is unknown.
- For screening purposes, in-house ELISA testing (e.g. Idexx 4DX that covers to *Ehrlichia* spp., *Anaplasma* spp., *Borrelia burgdorferi*, and *Dirofilaria immitis* antigen) are of value.
- ***Borrelia burgdorferi*** **(Lyme disease)** spirochete spread by *Ixodes* ticks. Cats appear resistant and most dogs appear to develop subclinical infection. Acute signs include fever, polyarthritis, lymphadenopathy, and anorexia. Chronic disease-causing shifting lameness. A few dogs develop severe progressive protein losing nephropathy (PLN).
  - Treatment with doxycycline for 10 mg/kg PO q24 hr for a month.
- ***Anaplasma phagocytophilium*** causes granulocytic disease transmitted by *Ixodes*. Clinical signs – fever, lethargy, inappetence, peripheral lymphadenopathy, and lameness.
  - Treatment with doxycycline for 10 mg/kg PO q24 hr for 2 weeks
- ***Ehrlichia canis*** tick-borne intracellular rickettsial parasite transmitted by the brown dog tick (*Rhipicephalus sanguineus*).
  - The acute phase (8–20 post infection) has non-specific signs – fever, anorexia, and lymphadenomegaly. Chronic infection leads to leucopenia, thrombocytopenia, and platelet dysfunction, leading to severe bleeding in some cases. Treatment with doxycycline for 10 mg/kg PO q24 hr for a month.
- ***Leishmania*** carried by sand flies, rarely other parasites. Follows a very chronic course, with classic signs including weight loss, lymphadenopathy, lameness, and exfoliative dermatitis. Diagnosis by identifying the parasite in lymph nodes or bone marrow, PCR, or antibodies. Treatment is protracted and of variable success.
  - Allopurinol in with meglumine antimonate or miltefosine.
- ***Babesia*** tick-borne carried by *Dermacentor* and *Rhipicephalus*. Can cause severe and life-threatening anaemia in dogs. Organism seen in the red cells or by PCR. Treatment usually with two injections of imidocarb, given at two-weekly intervals. Cases with severe haemolytic anaemia may require blood transfusions. Treatment depends on babesia species: e.g. imidocarb; azithromycin and atovaquone

3.15 ONCOLOGY

3.15.1 *Prevalence of neoplasia* 3.15.2 *Lumps* 3.15.3 *Required tests* 3.15.4 *Therapeutic modalities* 3.15.5 *Common chemotherapy drugs* 3.15.6 *Calculating the dose of chemotherapy agents* 3.15.7 *Dietary management of neoplasia* 3.15.8 *Paraneoplastic syndromes*

INTRODUCTION

3.15.1 PREVALENCE OF NEOPLASIA

- Approximately 30% of sick medical cases have neoplasia as a cause of their presenting signs.
- Neoplasia is the cause of death in 45% of dogs over ten years of age.

HISTORY AND PHYSICAL EXAMINATION

3.15.2 LUMPS

- Owners should be asked about lumps/bumps they have noticed as they groom or stroke their pet, as these can be significant and are easily missed during physical examination. The history should be augmented by careful physical examination, recording position and size of any masses where feasible.

DIAGNOSIS

3.15.3 REQUIRED TESTS

- Changes in routine haematology and biochemistry are variable depending on the site, type, and size of the tumour(s).
- Imaging is crucial for evidence of non-cutaneous mass lesions.
- Cytology/histology is required for definitive diagnosis.

! *The presence of a mass on palpation/imaging does not make that mass neoplastic, nor is it possible to palpate a skin lump to determine whether it is a lipoma.*

THERAPY FOR NEOPLASTIC DISEASE

3.15.4 THERAPEUTIC MODALITIES

- For many tumours the most effective therapy requires a multimodality approach.
- **Surgery** (see 4.3)
  - For diagnosis and treatment of localised neoplasia.
  - Can be curative, with appropriate margins.
  - Maximum information (tumour type, extent, spread) prior to a first surgery gives the best outcome.
  - Marking the edges of a removed tumour with tissue paints or variable suture numbers helps in assessing tumour-free margins.
  - Note that post-operative analgesic requirements can be high.
  - See also 4.3.4 – approach to a lump.
- **Radiation**
  - Primarily for treatment of localised tumours.
  - Side-effects are common – those seen depend on the area irradiated.
  - Limited availability apart from ^{131}I.
    - Dose needs to be fractionated, so repeat treatments required.
    - Multiple small fractions are more effective.
  - Delivery of radiation is by orthovoltage units, radioactive sources, or linear accelerator (most common in UK). The optimal source will depend on type and position of the tumour being treated.
  - Potential cases should be discussed with a radiation oncologist.
- ***Chemotherapy*** has been tried in virtually every tumour described. Many tumours have limited sensitivity. The published data are often limited as to the true increase in survival that chemotherapy can deliver.
  - Multi-agent treatment tends to be more effective than single agent.
  - Mild side-effects are common (40% of patients), severe side-effects are relatively rare.
  - Side effects occur most often 72–96 hours post treatment.
  - **Metronomic therapy** is repetitive, low-dose chemotherapy that aims to minimise toxicity.

- ***Other therapies***
  - COX-2 inhibitors, e.g. piroxicam, meloxicam, have been used in the management of transitional cell carcinomas. Their efficacy in other carcinomas is unknown.
  - Tyrosine kinase receptor inhibitors are an alternative to chemotherapy for non-resectable mast cell tumours but have been used in a wide range of other tumours with variable success. Side-effects can be significant.
  - Vaccines: xenogeneic DNA vaccine is available for certain melanoma types.
  - Other therapies include individualised T-cell therapy, photodynamic, antiangiogenic, and gene therapy.
- ***Immunotherapy*** is a rapidly expanding field in humans with some remarkable results and such treatments are beginning to impact veterinary cancer therapy.

! *Patients with lymphoma that have been treated with prednisolone prior to diagnosis may have shorter survival times.*

3.15.5 COMMON CHEMOTHERAPY DRUGS

- Suitably trained personnel and appropriate health and safety protocols should always be followed.
- Contaminated waste should be disposed of in specifically designated containers.
- Injectable drugs should be drawn up in a fume cupboard or using other safety devices, such as PhaSeal™ or use services that dispatch prefilled syringes and appropriate delivery systems.
- Certain staff should avoid being involved with chemotherapy.
- An overarching practice policy on chemotherapy use is helpful.
- Tablets should never be split – some drugs are available in different sizes via recompounding manufacturers, e.g. lomustine or cyclophosphamide.
- Good record keeping is essential in case of adverse events.
- ***Prednisolone*** is lymphocytolytic to neoplastic lymphocytes: initial dose rate 2 mg/kg or 50 mg/m^2. It may also reduce tumour-associated inflammation and, therefore, tumour size.
- ***Vincristine/vinblastine*** interferes with microtubule assembly. Used primarily to treat lymphoproliferative disorders and mast cell tumours. Always give via IV catheter.
  - *Vincristine dose for lymphoma*: 0.5–0.75 mg/m^2 maximum once weekly.
  - *Side-effects*: bone-marrow suppression usually mild; peripheral neuropathy; constipation; severe perivascular irritant.
- ***Cyclophosphamide*** crosslinks DNA preventing synthesis and function; used to treat lympho- and myeloproliferative and immune-mediated disease, and some sarcomas and carcinomas.
  - *Dose*: dependent on protocol.
  - *Side-effects*: myelosuppression (nadir 7–14 days); sterile haemorrhagic cystitis reduced by coadministration of furosemide; GIT upset; hepato- and nephrotoxicity; reduced hair growth.
- ***Chlorambucil*** is similar to, and a substitute for, cyclophosphamide but does not cause sterile cystitis. Used for management of small cell lymphoma in cats, and for chronic lymphocytic leukaemia. More commonly used as an immunosuppressive agent to treat immune mediated disease.
  - *Dose*: dependent on disease.
  - *Side-effects*: anorexia; nausea; vomiting; rarely, bone marrow suppression.

Table 3.15a Body weight/Body surface area

BWt (kg)	BSA (m2)	BWt (kg)	BSA (m2)	BWt (kg)	BSA (m2)
0.5	0.063	11	0.49	32	1.01
1.0	0.100	12	0.52	34	1.05
1.5	0.134	13	0.55	36	1.09
2.0	0.163	14	0.58	38	1.13
2.5	0.184	15	0.60	40	1.17
3.0	0.208	16	0.63	45	1.26
3.5	0.231	17	0.66	50	1.36
4.0	0.252	18	0.69	55	1.46
4.5	0.273	19	0.71	60	1.55
5	0.292	20	0.74	65	1.67
6	0.33	22	0.78	70	1.72
7	0.36	24	0.83	75	1.80
8	0.40	26	0.88	80	1.88
9	0.43	28	0.92	90	2.03
10	0.46	30	0.96	100	2.18

- ***Azathioprine*** acts to inhibit purine synthesis necessary for cell proliferation in leucocytes; used as an immunosuppressive agent; potentially highly toxic in cats.
  - Dose: in dogs, 2 mg/kg q24 hr until remission, then 0.5–2 mg/kg q48 hr.
  - Side-effects: bone marrow suppression can be severe in some dogs, due to deficiency of an enzyme required for metabolism.

3.15.6 CALCULATING THE DOSE OF CHEMOTHERAPY AGENTS

- Most chemotherapeutic agents have a relatively narrow therapeutic window.
- Drug dosing by body weight tends to underestimate the dose in small patients and overestimate in large ones. Body surface area (BSA) is closer to the metabolic ability. See Table 3.15a.
- Adjustment should be made for obese or ascitic patients and a 'guesstimate' made of their ideal weight, or weight without fluid.

NUTRITION

3.15.7 DIETARY MANAGEMENT OF NEOPLASIA

- Maintaining body weight improves survival times in patients with neoplasia.
  - Omega-3 polyunsaturated fatty acids reduce tumour necrosis factor-α, which can be associated with weight loss.
  - Inappetence can be a major factor, so highly palatable energy-dense diets are recommended.
  - Energy is best provided as fats. Energy requirement can be twice that of a similarly sized animal without neoplasia.
  - Protein levels should be increased at 30–45% (dog), 35–45% (cat) of metabolisable energy. High-dose arginine supplementation (>2% on dry matter basis) may be helpful.
- A wide variety of other food ingredients and nutraceuticals have been recommended for cancer patients, but evidence of efficacy is lacking.

SELECTED TOPIC

3.15.8 PARANEOPLASTIC SYNDROMES

- These are disease states that occur secondary to the metabolic, endocrine, or haematologic effects of a tumour.
- These effects can be distant from the tumour itself and be responsible for the clinical presentation, e.g. hypercalcaemia associated with an anal sac carcinoma, causing a presentation for PU/PD.
- ***Haematologic***
  - Anaemia due to chronic disease, bone marrow invasion, immune-mediated or microangiopathic haemolysis, GI and external loss, hypersplenism.
  - Thrombocytopenia and altered coagulation (DIC).
  - Hyperviscocity due to monoclonal gammopathy, polycythaemia.
  - Leucocytosis and eosinophilia – rare.
- ***Metabolic and endocrine***
  - Cancer cachexia involves anorexia, weight loss, fatigue, and immune dysfunction; maintenance energy requirement can double in cancer cases. Cancer cachexia is caused by alterations in carbohydrate, protein and lipid metabolism, and it decreases survival times.
  - Fever induced by cytokines accounts for about 10% of cases presenting with PUO (2.12).
  - Hypercalcaemia due to release of parathyroid-related peptide (PTH-rP).
  - Hypoglycaemia is observed with insulinomas, but also liver, pulmonary and muscle tumours. It can be secondary to sepsis, e.g. GI tumour rupture.
  - Hyperhistaminaemia: release from mast cells causing local oedema, erythema and pruritus and distant GI ulceration.
- ***Neuromusculoskeletal***
  - Demyelination and axonal degeneration; there is probably an underlying autoimmune mechanism.
  - Myasthenia gravis; usually secondary to thymoma.
  - Hypertrophic pulmonary osteopathy (Marie's disease).
- ***Dermatologic***
  - Hepatocutaneous syndrome.
  - Alopecia (cats).
  - Exfoliative dermatitis (in cats with thymoma).
  - Small, hyperplastic, dermal collagenous nodules.
  - Lung digit syndrome.

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4

ANAESTHESIA, ANALGESIA, AND SURGERY

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4.1 ANAESTHESIA

4.1.1 *Introduction* 4.1.2 *Sedation and premedication* 4.1.3 *Use of induction agents* 4.1.4 *Maintenance of anaesthesia* 4.1.5 *Anaesthetic circuit choice* 4.1.6 *Monitoring*

4.1.1 INTRODUCTION

- For any procedure, the choices of sedation or anaesthesia, and of which agent(s) to use are very situation dependent. For a particular case consider:
  - The patient's age, breed, and temperament.
  - How still the patient is required to be.
  - The pain and duration of the procedure.
  - Whether a secondary procedure may become necessary.
  - Primary and intercurrent disease (if any).
- Heavy sedation with α2-agonists can cause profound cardiorespiratory depression. Light anaesthesia is often safer than heavy sedation.
- In some circumstances, local or regional anaesthesia should be considered, usually in combination with sedation or general anaesthesia (GA).

4.1.2 SEDATION AND PREMEDICATION

- Sedative choice will depend on the individual patient's temperament, procedure being undertaken, and underlying disease.
- Examples of protocols in routine use, with suggested dose rates, are shown in Table 4.1a. The drug's effect is generally more reliable if given by IM rather than SC injection.
- Use lower doses for light sedation, as a premedicant, or in sick patients.
- Some patients (especially cats) benefit from mild sedation prior to travelling to their appointment, this can have a marked impact on the ease of examination, handling, and performing minor procedures such as blood sampling or ultrasound. Gabapentin (20–30 mg/kg) or tramadol (6–10 mg/kg) is given 1–2 hours before appointment.

4.1.3 USE OF INDUCTION AGENTS

- Induction (Table 4.1b) should induce rapid, safe and stress-free unconsciousness.
- Induction should be sufficient to allow intubation and to allow the maintenance protocol to become effective.
- Intubation maintains an open airway, provides oxygen, and protects against aspiration of refluxed fluid.

Practical tips

- Don't forget to pre-oxygenate where appropriate.
- To smooth the induction and reduce the dose of induction agent required in critically ill patients, give diazepam emulsion (0.2–0.5 mg/kg) or midazolam (0.2–0.3 mg/kg) IV 20–30 minutes after premedication and before induction.

4.1.4 MAINTENANCE OF ANAESTHESIA

- Anaesthesia can be maintained by CRI or intermittent bolus injection of propofol or alfaxalone. Ideally a tube should be placed and oxygen given.
- The majority of anaesthetics are maintained by volatile gaseous agents.
  - Isoflurane and sevoflurane (which gives quicker induction and recovery) are used most widely.
    - Isoflurane maintenance for around 50% of individuals can be achieved at 1.5%; sevoflurane at 2.5%.
  - Both can be used as induction agents (3–5% and 6–8%, respectively) with suitable scavenging. Induction is unpredictable and can be stressful if the gas is delivered by mask.

Table 4.1a Sedatives and premedicants

Agent and dose rate	Comments
ACP (0.01–0.02 mg/kg) + buprenorphine (0.01–0.02 mg/kg)	Light sedation for minor, non-painful procedures, e.g. ultrasound or premedicant for mild–moderately painful surgery ± NSAID
ACP (0.01–0.02 mg/kg) + butorphanol (0.1–0.3 mg/kg)	More profound sedation than buprenorphine, premedicant for mildly painful procedures
ACP (0.01–0.02 mg/kg) + methadone* (0.1–0.3 mg/kg)	Sedation similar to butorphanol but significantly better pain relief for moderate- to markedly-painful procedures
Medetomidine** (10–20 μg/kg [dog]) (15–30 μg/kg [cat]) with opioid ± ACP***	10 μg/kg dose is useful for sedating cats. Mild to profound dose-dependent sedation. Level of pain relief associated with opioid use
Alfaxalone in cats (1.5–2 mg/kg) IM + opioid + midazolam	Good levels of sedation for most procedures Good safety margin for cats with cardiovascular disease
Midazolam (0.25–0.5 mg/kg) + ketamine (2.5–5 mg/kg)	Primarily in cats. Can be given IM or use 50:50 dose combination to effect IV. Level of sedation unpredictable
Benzodiazepine (0.25–0.5 mg/kg) + opioid	Not for general use but relatively safe in critically ill animals. Give opioid first then benzodiazepine IV 20–30 minutes later

* can use pethidine 2–10 mg/kg instead. NB IM only not IV

** dexmedetomidine given at half dose rate (same volume)

*** valuable addition in nervous or aggressive patients

Dexmedetomidine (15–40 μg/kg) or medetomidine (20–80 μg/kg) can be used at higher doses as a single agent for sedation but combination are safer and more commonly used

Reversal of sedation

Atipamezole reversal agent for α2-agonists is given at equal volume (dog); half volume (cat) of medetomidine or dexmedetomidine used in the original sedation.

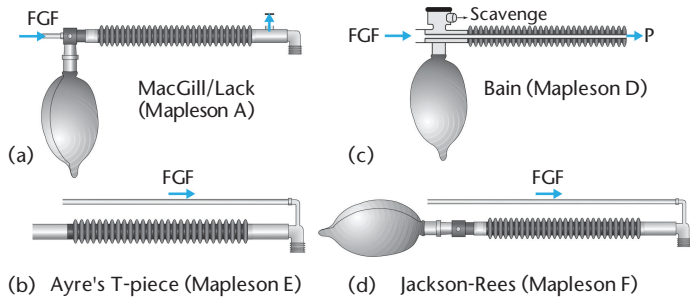
4.1.5 ANAESTHETIC CIRCUIT CHOICE

- Rebreathing circuits with carbon dioxide absorption (by soda lime). They use much lower flow rates of oxygen and smaller amounts of volatile agent. The Humphrey ADE circuit, for example, is a multipurpose system:
  - Use without soda lime for dogs and cats weighing 7–10 kg, with fresh gas flow (FGF) rate of 90–130 ml/kg/min to prevent rebreathing.
  - Use with soda lime for heavier dogs, with FGF rate of 100 ml/kg/min for 5 minutes after induction reducing to 10–20 ml/kg/min for maintenance (minimum flow 0.5–1 l/min).
- Non-rebreathing circuits are MacGill/Lack, T-piece, Bain, and Jackson-Rees (Figures 4.1a, b, c, and d). They are chosen on the basis of patient size.
- FGF rates are much higher:
  - T-piece, Jackson Rees, and Bain: 250–300 ml/kg/min.
  - Lack and MacGill: 150 ml/kg/min.
- 0.5, 1, 2, 3, and 5-litre bags are commonly used in veterinary practice: choose a bag that is approximately 0.1 l/kg body weight.

Table 4.1b Induction agents

Drug	Propofol	Alfaxalone	Medetomidine + ketamine + opioid
Use	Standard IV induction agent	Standard IV induction agent	Combination IM induction agent
Dose rate	Start with 1 mg/kg in cat and dogs Titrate to effect until intubation possible With premedication 1–4 mg/kg (dog); 2–5 mg/kg (cat) Maintenance CRI 0.1–0.4 mg/kg/min	Give slowly to effect With premedicant up to 2 mg/kg (dog); 5 mg/kg (cat) Maintenance CRI 6–9 mg/kg/hr Can be a useful IM agent in cats	Ketamine: 1.25–5 mg/kg Medetomidine: 25–80 µg/kg Dexmedetomidine: 12–40 µg/kg Buprenorphine: 0.01–0.02 mg/kg Butorphanol: 0.1–0.3 mg/kg Methadone: 0.1–0.3 mg/kg
Formulation	Shake well before using 10 mg/ml lipid emulsion, no preservative so discard at the end of the day 10 mg/ml non-lipid emulsion, shelf life 28 days from opening Contains benzyl alcohol – do not use as an infusion as benzyl alcohol accumulates and is neutoxic	Pregnane neurosteroid 10 mg/ml solution solubilized in cyclodextrin. Shelf life once broached depends on product	Ketamine: 100 mg/ml Medetomidine: 1 mg/ml Dexmedetomidine: 0.5 mg/ml Buprenorphine: 0.3 mg/ml Butorphanol: 10 mg/ml Methadone: 10 mg/ml
Comments	Rapid induction causes apnoea, cyanosis, bradycardia and severe hypotension. Muscle rigidity, paradoxical movement and tremors in dogs on induction may worsen with further doses of propofol; they wane with time. Repeat doses can be associated with Heinz body anaemia (cat)	Do not use in combination with other IV anaesthetic agents. Similar dose-dependent cardiorespiratory effects to propofol. Metabolized in liver and excreted via bile and kidneys. Half-life 25 min (dog); 45 min (cat). Longer recovery time required if CRI or top-up boluses used for maintenance	Use higher end of dose range for cats and reduce if the cat is sick. Atipamezole can be used as a partial reversal agent but careful dose calculations are still required Tip: use only one or two combinations to become familiar with their effect Reversal: Do not use until at least 30 minutes after (dex)medetomidine; reduce dose to 50% if >60 minutes. Reversal not helpful after 2 hours

✪ Using a heat and moisture exchange (HME) can reduce the cooling effect of inhaling gaseous anaesthetics.



Figures 4.1 (a) MacGill/Lack (Mapleson A), (b) Ayre's T-piece (Mapleson E), (c) Bain (Mapleson D), (d) Jackson-Rees (Mapleson F)

Table 4.1c Anaesthetic monitoring methods

	Blood pressure (BP)	**ECG**	**Pulse oximetry (SpO_2)**	**Capnography ($ETCO_2$)**
Information obtained	Doppler: systolic BP. Oscillometric: systolic, diastolic, and mean BP	Heart rate and rhythm	Oxygen saturation of haemoglobin in arterial blood and pulse rate	Partial pressure of CO_2 in expired gas
Strengths	Assessment of pulse rate and quality of output	Visual and numerical display. Essential to distinguish poor circulation due to arrhythmia vs. circulatory collapse	Simple and easy to use. Can be placed at a variety of locations	Reflects interaction of metabolism, ventilation and pulmonary circulation, as well as functioning of anaesthetic equipment
Limitations	Can be difficult to place, particularly on sick patients that are vasoconstricted with poor pulse pressures	No information about the quality of circulation. If T waves are tall the machine will often double count	Vasoconstriction and movement reduce reliability. Oxygen dissociation curve is sigmoid so SpO_2 slow to respond to a fall in arterial oxygenation. Hypoventilation can exist with high SpO_2 if O_2 is being supplemented	Limited value without capnographic curve. Experience required to interpret curve and decide upon appropriate action

4.1.6 MONITORING AN ANAESTHETIC

- Good observational monitoring of anaesthesia is vital, especially in sick patients (Table 4.1c).
- Manual counting of heart, pulse, and respiratory rates serves to check machine values.
- Pulse quality and mucosal colour additionally evaluate the circulation.

- Regularly monitor rectal temperature.
- Single measurements are of relatively limited value; trend monitoring is much more useful.

4.2 ANALGESIA

4.2.1 *Introduction and assessment of pain* 4.2.2 *Opioid analgesics* 4.2.3 *CRI pain relief* 4.2.4 *Non-steroidal anti-inflammatory drugs* 4.2.5 *Reversal and management of overdose* 4.2.6 *Other approaches to pain relief*

4.2.1 INTRODUCTION AND ASSESSMENT OF PAIN

- Non-steroidal anti-inflammatory drugs (NSAIDs) are the mainstay for chronic pain relief in cats and dogs.
- Acute pain relief, especially in haemodynamically compromised patients, is often better delivered with opioids which can be used in combination with NSAIDs.

Assessment of pain

An expectation that a procedure or disease will be painful based on human experience, is not unreasonable, it does not take into account individual patient perception. Pain scoring systems are now routinely used but should not be the sole assessment. If in doubt, analgesia should be given and patient response observed.

Recognition and assessment of pain is difficult in animals leading to systems based on physiologic (heart, respiratory rate) parameters behaviour and response to palpation.

Using pain scales has value; consistency can be improved by training and scoring by the same individual. A variety of scales are used depending on whether pain is acute, e.g. Glasgow composite pain scoring scale or chronic where quality of life is also a factor and pain scales may be specific for a disease process, e.g. osteoarthritis – canine brief pain inventory.

4.2.2 OPIOID ANALGESICS

- Opiates deliver relatively safe, low toxicity, effective pain relief. Many are controlled drugs.
- Table 4.2a outlines the use of commonly prescribed opioid products; the majority are not licensed for use in cats and dogs.
- Sedative effects of opioids are more prominent in dogs than cats, cats get a state of euphoria and if dose is excessive, excitement.
- Dysphoria can occur in both dogs and cats if doses are too high.
- All opioids will cause some respiratory depression.
- The duration of action and dose required can be quite variable between individuals. The dose required is usually lower in patients with hepatic disease.

  A number of opioids can be given as epidural pain relief, or as continuous rate infusion (CRI) solutions. The latter are best in combination (4.2.3).

4.2.3 CRI PAIN RELIEF

- CRI cocktails are intended to follow on from loading-dose pain relief. Two useful formulations are:
  - Morphine, lidocaine, and ketamine (MLK): 60 mg M + 1000 mg l + 60–120 mg K/l (0.9% NaCl) @ 1–3 ml/kg/hr.
  - Fentanyl, lidocaine, and ketamine (FLK): fentanyl instead of morphine in the above MLK: 2 mg F/l (0.9% NaCl) @ 1–3 ml/kg/hr; protect bag from light.

Table 4.2a Opiod Analgesics

Generic name	Site of action	Route of administration*		Dose rate (mg/kg)		Legal category/comments
		Dog	Cat	Dog	Cat	
Buprenorphine	Partial agonist MOP receptor	IV, IM, SC	IV, IM, SC, transmucosal	0.01–0.02 q6hr	0.01–0.02 q6hr	S3. Absorbed orally in cats (oral pH different to man/dogs), use injectable solution rather than sublingual tablets
Butorphanol	Primarily KOP agonist, MOP antagonist	IM, SC (10/ml) PO (5, 10)		0.2–0.5 q6–12hr	0.2–0.5 q6–12hr	POM-V
Codeine	Mechanism unclear, MOP receptor?	PO (15, 30, 60; 5/ml)	PO (15, 30, 60; 5/ml)	0.5–2 q12hr	0.5–2 q12hr	POM-V. Also used as cough suppressant and anti-diarrhoeal. Analgesic effect is mild. Injectable preparation available
Fentanyl	Pure MOP agonist	IV (0.05/ml); patch (12, 25, 50, 75, 100 μg/hr)		patch (4 μg/kg/hr) or as FLK infusion		S2
Methadone	Pure MOP agonist	IV, IM (10/ml)	IV, IM, SC (10/ml)	0.1–0.5 IM q4–6hr	0.1–0.3 IM q4–6hr	S2
Morphine	Pure MOP agonist	IV, IM (usually 10/ml)	IV, IM (10/ml)	0.5 IM q3–4hr	0.1–0.4 q3–6hr	S2. Oral tablets, suppositories, syrups available; will often cause vomiting. Injectables 10, 15, 20, and 30 mg/ml. *Check concentration especially where practice also uses morphine in horses!*
Pentazocine	Weak KOP, antagonistic MOP	po (25, 50)	Not advised	2–6 q3–4hr	–	S3
Pethidine (= meperidine)	Pure MOP agonist	IM, SC (10–50)	IM, SC (10–50)	2–10 q1–2hr	5–10 q1–2hr	S2
Tramadol	All opioid receptors, esp. MOP; weak agonist	po (50, 100, 200, 300, 5 / ml)	PO	2–5 q12hr	2–4 q12hr	POM. Also inhibits noradrenaline and 5HT pathways, thus providing alternative pathways for pain relief

* Text in parentheses are injectable/oral solution strength (mg/ml) or tablet size (mg)
Legal categories: POM-V = prescription-only medicine (veterinary); S2 = schedule 2; S3 = schedule 3

4.2.4 NON-STEROIDAL ANTI-INFLAMMATORY DRUGS

- NSAIDs moderate pain by blocking a variety of pathways (COX, LOX, and thromboxane).
- Table 4.2b outlines the use of commonly prescribed NSAIDs.
- A variety of other NSAIDs have been used in dogs and cats, but their use is difficult to justify, given the wide range of licensed products available.

4.2.5 REVERSAL AND MANAGEMENT OF OVERDOSE

- ***Misoprostol*** (Cytotec, 200 µg tablets): protects against NSAID-induced gastric ulceration. Can cause abdominal pain, diarrhoea, nausea, vomiting, and abortion. Dosage is 2–7.5 µg/kg q8–12 hr (dog), 5 µg/kg q8 hr (cat).
- ***Naloxone***: competitive antagonist for opioid receptor; use in accidental overdose or poisoning; 0.015–0.04 mg/kg IV, IM and SC.
- ***Atipamezole***: see Table 4.1b

4.2.6 OTHER APPROACHES TO PAIN RELIEF

- ***Anti-nerve growth factor monoclonal antibodies:*** are monthly injections for use in the management of osteoarthritis in dogs (bedinvetmab) and cats (frunevetamab). Response varies between patients but can provide effective, safe and convenient chronic pain relief.
- ***Gabapentin***: used for neuropathic and oncologic pain, mechanism of action is unknown. Dose up to 10 mg/kg PO q8 hr. Can cause mild sedation and ataxia.
- Benzodiazepines are used as muscle relaxants.
- ***Methocarbamol*** (Robaxin): skeletal muscle relaxant. Dose 20–45 mg/kg PO q8 hr. Side-effects: salivation, vomiting, lethargy, weakness, ataxia, and CNS depression.
- ***Amantadine***: 3–5 mg/kg PO q24 hr (dog), 1–4 mg/g PO q24 hr (cat)***, memantine*** 0.3–0.5 mg/kg PO q12 hr (dog): N-Methyl-0-aspartate- (NMDA-) antagonist analgesic.
- ***Amitriptyline***: 1–2 mg/kg PO q12–24 hr (dog), 1 mg/kg PO q24 hr (cat), NMDA-antagonist analgesic (also used for behavioural therapy, FLUTD and management of ureteroliths).
- **Non-drug modalities**: the value of physiotherapy, acupuncture and other therapies should not be underestimated.

✪ Early aggressive pain management reduces the 'wind-up' phenomenon and therefore the overall amount of pain relief required. In chronic cases, pain relief may actually reduce the rate of disease progression.

4.3 SOFT-TISSUE SURGERY

4.3.1 *Introduction* 4.3.2 *Minimising surgical infection* 4.3.3 *Haemostasis* 4.3.4 *Approach to a lump* 4.3.5 *Sutures* 4.3.6 *Bandages and dressings* 4.3.7 *Wound management* 4.3.8 *Post-operative care*

4.3.1 INTRODUCTION

- It is not possible within a text of this length to cover individual surgical procedures. The following is designed to address surgical issues that are common to most procedures.

! Surgery should be avoided if you are unsure of the technique, if you have no 'plan B' if your expected findings are not evident, or if there is inadequate post-operative/overnight care.

Table 4.2b Non-steroidal Anti-inflammatory Drugs (NSAIDS)

Generic name [Trade name]	Available preparations	COX selectivity	Comments	Administration route		Dose rate	
				Dog	Cat	Dog	Cat
Aspirin [various]	75, 300 mg (T)	Non-selective COX inhibitor	H, 1, 2	PO	PO	10–20 mg/kg q12hr	10–20 mg/kg q48–72hr
Carprofen [Rimadyl*]	20, 50, 100 mg (T) 50 mg/ml (IS)	Preferentially inhibits COX-2	H, 1, 3	IV, SC	IV, SC, PO	Initially 4 mg/kg, then 2 mg/kg q24hr PO	
Cimicoxib [Cinalgex]	8, 30, 80 mg (T)	Preferentially inhibits COX-2	H 1, 3	PO	Not authorised	2 mg/kg q24 hr	N/A
Cincophen [PLT]	200 mg (T) with 1 mg prednisolone	Uncertain	H 1 – ulcerogenic potential high	PO	Do not use	12.5 mg/kg q12 hr	N/A
Etodolac [EtoGesic*]	150, 300 mg (T)	Preferentially inhibits COX-2	H, 1, 3, 4	PO	Not advised	10–15 mg/kg q24hr	_
Firocoxib [Previcox]	57, 227 mg (T)	Preferentially inhibits COX-2	H, 1, 3	PO	Not advised	5 mg/kg q24hr	_
Grapiprant [Galliprant]	20, 60, 100 mg (T)	EP4 receptor antagonist	H, HR, 1, 3	PO	Not authorised	2 mg/kg q24 hr	N/A
Ketoprofen [Ketofen]	5, 20 mg (T) 1 mg/ml (IS)	Non-selective COX inhibitor	HR, 1,2, 3, 8	IV, IM, SC, PO	IV, IM, SC, PO	Initially 2 mg/kg, then 0.25 mg/kg q24hr	First dose 2 mg/kg then 1mg/kg q 24hr for 5 days
Mavacoxib [Trocoxil]	6, 20, 30, 75, 95 mg (T)	Preferentially inhibits COX-2	1, 3	PO	Not advised	2 mg/kg, 2nd dose @14d then monthly	_

(*Continued*)

Table 4.2b (Continued) Non-steroidal Anti-inflammatory Drugs (NSAIDS)

Generic name [Trade name]	Available preparations	COX selectivity	Comments	Administration route		Dose rate	
				Dog	Cat	Dog	Cat
Meloxicam [Metacam*]	1, 2, 5 mg (T) 0.5, 1.5 mg/ml (OL) 2, 5 mg/ml (IS)	Preferentially inhibits COX-2	H, 1, 3	SC, PO	SC	Initially 0.2 mg/kg, then 0.1 mg/kg q24hr	0.3 mg/kg (single) or initially 0.1 mg/kg then 0.05 mg/kg q24hr
Paracetamol [various]	120, 400, 500 mg (T) 25, 50 mg/ml (OL) 10 mg/ml (IS)	Antipyretic and analgesic via COX	H 1, 3, 7, 9	PO	Not advised	10 mg/kg q12hr	Toxic to cats
Piroxicam [various]	10, 20 mg (T) + 20 mg dissolvable	Therapy for adenocarcinoma	HR, 1, 3, 5	PO	No data	0.3 mg/kg q24hr	–
Robenacoxib [Onsior]	5, 6, 10, 20, 40 mg (T) 20 mg/ml (IS)	Preferentially inhibits COX-2	HR 1, 3	SC, PO	SC, PO	Initially 2 mg/kg, then 1 mg/kg q24hr	
Tolfenamic acid [Tolfedine]	6, 20, 60 mg (T) 40 mg/ml (IS)	COX selectivity uncertain	HR, 1, 2, 3	SC, IM, PO	SC, PO	4 mg/k q24hr for 4 days	4 mg/kg q24hr for 3 days

* Also produced by other manufacturers under different trade names

T = tablets; IS = Injectable solution; OL = oral liquid; COX = cyclooxygenase; H = hepatic metabolism with biliary excretion; HR = hepatic metabolism with renal excretion

List based on products available in UK – other products/formulations marketed in other countries; trade names may vary.

1. Standard GI and hypotension, lowest effective dose
2. Lower dose as anticoagulant or has anti-platelet action
3. Small risk of precipitating CHF in patients with cardiovascular disease
4. Other sizes available for people
5. Value of Piroxicam over meloxicam unclear, efficacy so far only been documented for transitional cell carcinoma of bladder
6. 5-lipoxygenase inhibition may be associated with an improved GI safety profile
7. Injectable preparation for IV use available for people, limited experience in dogs
8. Dogs' dose schedule can be repeated weekly
9. Little anti-inflammatory activity

4.3.2 MINIMISING SURGICAL INFECTION

Environment

- Use the operating theatre only for surgery.
- Clean every morning and evening (between every surgery if possible).
- Ideally, prepare patients (e.g. clipping, cleaning, anaesthetising) in a separate room.
- Ensure adequate lighting and, ideally, filtered, positive pressure ventilation, especially for advanced orthopaedics.
- Plan the surgical list: cleanest surgery first, finishing the end of the list with the infected or 'dirty' surgeries.

Preparation of the patient

- Clip a wide area that allows for movement of skin into the surgical field during closure.
- Vacuum or brush the loose hairs off the coat and cover feet with a waterproof drape to prevent strikethrough.
- Use antiseptic scrub for skin preparation (chlorhexidine/povidone iodine), then alcohol-based antiseptic spray.

Preparation of the surgeon

- Use a surgical cap and, ideally, a mask. Talking increases the risk of aerosol infection!
- Use a standard protocol to scrub hands, nail beds, arms; this takes 5 minutes.
- Use sterile gloves and gowns for every surgery.
- Change clothes used for consulting, house visits, or other procedures into clean scrubs and shoes for the operating area.

Surgical technique

- Drape the whole animal and cover the table and trolley; water-resistant drapes prevent strikethrough.
- If a surgery is difficult, make the incision longer and get an assistant to scrub in.
- ***Swabs*** dampened with sterile saline, for covering tissues and skin edges during long surgery, prevent desiccation.
    - Use swabs with x-ray-detectable threads for cavity surgery.
    - Count your swabs onto the trolley and out of the animal.
- ***Contamination***: if tissues become contaminated during surgery, lavage the surgical site with sterile saline prior to closure.
    - Warm sterile saline (large volumes) is needed for abdominal lavage; remove with suction prior to closure.
- If the site is contaminated (e.g. strikethrough of drapes, GIT surgery), change gloves and drapes prior to closure.
- Infected or contaminated surgery: change instruments for closure; don't re-use suture materials used on GI/urinary tract.

4.3.3 HAEMOSTASIS

- Haemostasis is important, especially in invasive or complex surgery, in order to achieve:
    - Shorter surgery times.
    - A 'dry' surgical field, thereby improving visibility and reducing the risk of iatrogenic injury.
    - Reduced post-operative morbidity due to blood loss or hypotension.
    - Less risk of unidentified bleeding after abdominal or thoracic surgery.
    - Improved tissue healing, reduced post-operative oedema, and less risk of infection.
    - Reduced post-operative mortality and re-operation rates.

- Whole blood and blood products are expensive and in relatively short supply and are not a substitute for haemostasis.
- ***Ligatures***: suture material should be small gauge with good knot security and handling characteristics – preferably a synthetic absorbable material.
- The surgeon should be familiar with the techniques used to place ligatures without the throws on the knot locking prior to tightening around the pedicle.
    - The sliding knot is used to place a secure ligature, deep within a body cavity, but the tissue incorporated in the knot should be carefully examined prior to tightening.
    - One-handed ties are also used to place secure ligatures on vessels in awkward positions.
- ***Haemostatic equipment*** to seal small vessels during surgery includes:
    - Electrosurgery (monopolar or bipolar).
    - Laser.
    - Advanced electrosurgery (i.e. Ligasure, Enseal, etc.) and ultrasound (i.e. harmonic scalpel).
- ***Promotion of clotting***: in order to seal multiple small vessels or vessels that are difficult to access (e.g. friable surfaces [nasal turbinates, liver], bone surfaces, abraded skin/wound surfaces) techniques can be used to augment natural clotting. The method chosen must not delay healing or cause damage to adjacent structures. Two possibilities are:
    - Reduce blood flow to the affected area allowing clot formation, using:
        - Tourniquet and pressure – temporarily, for first aid or intra-operatively. Minor haemorrhage should stop after 2–3 minutes.
        - Cold packs or cold sterile saline (reduce blood flow).
        - Topical vasoconstrictors (e.g. dilute chilled adrenaline).
    - Topical haemostatic agents, (like bovine collagen, gelatin, cellulose, and other plant-based polymers) majority not licensed for use in animals, provide local tamponade and a scaffold to help clotting and are usually removed carefully once successful; precise data on how they work can be lacking. Useful to enhance clotting of friable surfaces and other situations where ligation or heat is not possible/appropriate.
    - Autogenous muscle also contains tissue factor, making it an active haemostatic agent.
    - Systemic drugs to reduce blood pressure (acepromazine) – use with extreme care.
    - Systemic pro-coagulants; tranexamic acid is being used more widely to manage significant haemorrhage (see Section 5.2).

4.3.4 APPROACH TO A LUMP

Assessment and planning

- All masses that are affecting the patient (or likely to soon), growing, or changing, should be investigated and not 'watched'.
- Not all lumps are neoplastic (see 3.15).
- For tumours before surgery, identify the tumour type and anatomy of the primary tumour, grade (tissue biopsy) and stage (metastasis (TMN)).
- Perform with minimal morbidity.
- Aim for cure in the first surgery; debulking is rarely indicated.
- Incompletely resected tumours are more likely to recur.
- Consider whether appropriate adjuvant therapy, e.g. radiation, chemotherapy, or immunotherapy is indicated.
- Know when not to operate (when surgery is of no ultimate benefit to the patient), e.g. the presence of significant residual disease despite surgery.

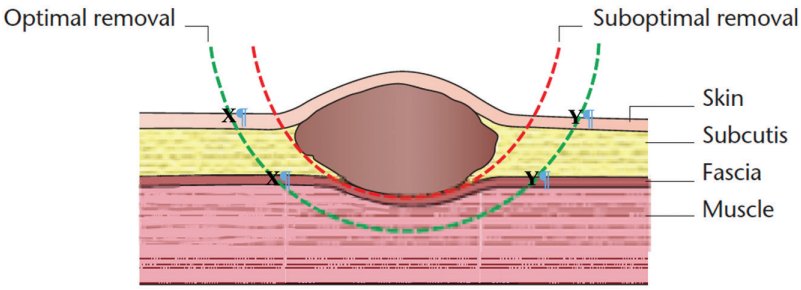


Figure 4.2 **Soft-tissue tumour excision.** In suboptimal removal (tumour peeled off fascia), microscopic disease may remain. In optimal removal, surgery should be curative.

Tumour staging

- Ascertain primary tumour type and grade. The most common superficial canine tumours are lipomas, mast cells, or soft-tissue sarcomas.
- Fine-needle aspiration (FNA) is useful for investigating lipoma, lymphoma, and mast-cell tumour; a non-diagnostic FNA suggests non-neoplastic disease or a sarcoma, so repeat FNA or obtain a biopsy.
- Tru-cut or incisional biopsy (unless wide excision biopsy is straightforward) if FNA is non-diagnostic.
- Lymph nodes: palpation alone is inaccurate. Consider FNA, biopsy, or excision if there is lymphadenopathy.
- Look for distant metastasis (thorax, abdomen) using radiography, ultrasound, or CT/MRI.

Principles of oncologic surgery

- Plan the extent of resection to remove the palpable tumour and a surrounding area of normal tissue, to ensure removal of all tumour cells (Figure 4.2). Higher-grade tumours require a wider area of resection.
- A lateral skin margin of 1–5 cm (depending on tumour type and grade) and a deep fascial layer are mandatory. The deep margin is the most common margin for incomplete excision.
- Consider suturing the skin to the deep fascia around the tumour (X to X and Y to Y in Figure 4.2) as you incise these, to enclose the mass inside a tidy parcel of skin and fascia, reducing cancer contamination of the wound bed.
- Removing masses without FNA or biopsy may lead to choosing inappropriate margins of resection. 'Shelling out' will only remove the grossly visible tumour tissue and recurrence is likely except with lipomas.
- Closure after tumour resection can be by second intention, or can be primary intention, which may require advanced surgical techniques. Early referral for definitive treatment is likely to have a better outcome than referral after resection with dirty margins.

4.3.5 SUTURES

Suture materials

- Synthetic suture materials are preferred to organic suture (catgut) as they are removed by hydrolysis, leading to a gradual and predictable loss of tensile strength. The suture should be at least as strong as the target tissue (skin/fascia > intestines > bladder) and lose tensile strength at a similar rate to the tissue gaining it.
- Monofilament sutures have minimal drag and form tight knots but are difficult to handle. Coated monofilament sutures are easier to handle but may potentiate infection and will 'drag' through tissue.

- ***Absorbable*** synthetic suture is generally used other than for skin sutures. A suture with prolonged tensile strength and slow absorption is appropriate for most situations, especially where early tissue separation could be problem, e.g. linea alba, blood vessel ligatures, intestines.
    - Examples are polydioxanone (PDS, Ethicon) and polyglyconate (Maxon, Covidien).
- ***Subcuticular*** sutures can have more rapid loss of tensile strength and absorption. Example: poliglecaprone (Monocryl, Ethicon).
- ***Permanent*** synthetic suture is used for skin or when suture absorption is problematic, e.g. perineal rupture repair. Example: nylon (Monosof, Covidien).

Suture size

- Suture size selection depends on the size of animal and tissue to be sutured. Large-gauge suture is to be avoided, as knots are less secure. Appropriate size selection would be:
    - Skin/subcuticular tissue: 1.5–2 metric (4/0–3/0 USP).
    - Linea alba: 3–3.5 metric (2/0–0).
    - Vascular pedicles, e.g. ovary: 3–3.5 metric (2/0–0).
    - Intestines: 1.5 metric (4/0).

Suture technique

- Swaged-on needles are preferred. Reverse cutting needles used for skin, but tapercut or taperpoint needles for viscera and fascia.
- The knot is the weakest part of a suture. A minimum of 4 throws are required for polydioxanone and nylon, with 1 and 2–3 extra throws, respectively, at the beginning and end of a continuous suture. A square or surgeon's knot is preferred and each throw must be securely tightened.
- Ligatures must be placed tightly, which may be aided with the use of a sliding/'slip' knot using monofilament suture material.
- Appositional sutures in incisions should be placed so that the tissues are held in snug apposition without strangulation.
- An Aberdeen (self-locking) knot is a useful way to bury the end of a continuous subcuticular suture.
- Note that failure of a suture line is usually related to surgical technique, rather than the choice of suture material or size for example taking adequate bites of tissue to avoid pull-through (dehiscence).

4.3.6 BANDAGES AND DRESSINGS

- Wounds are dressed with a primary (contact) layer followed by a secondary layer (padding and absorption) and finally a tertiary (protective) layer.
- ***Primary layers*** Assess the wound and determine the effect you want for the contact layer – protective, debriding, or actively promoting healing. Different dressings might affect how the wound looks but have no clinical impact on healing.
- ***Secondary layers*** Use synthetic or cotton wool for absorption, with an extendable, elastic, non-adherent bandage (e.g. TreatRap or equivalent) to secure the padded layer.
    - Wrap evenly, with about 50% overlap of each turn of the bandage. Depending on the level of support, this layer may be only 2–3 turns thick, or very heavy (e.g. a Robert Jones bandage).
    - The natural contours of the limb should be evened out, so that the bandage is roughly the same diameter all the way along; this reduces the risk of bandage injury. Use padded 'donuts' over bony prominences like the point of the hock or elbow, pad under the dew claw and between the digits, use stirrups to prevent bandage slipping.

- ***Tertiary layers***
  - Use a self-adherent, water-resistant material (e.g. Vetrap®).
  - Apply with even tension – not too tightly – up and down the limb.

! Bandage injuries can cause massive tissue loss, amputation, or death!

! Complications are caused by the bandage slipping or being applied too tightly or unevenly. The patient may chew at the dressing within 1–2 hours of application if it is causing restriction of circulation.

! All owners should be provided with written discharge instructions on bandage care, including instructions to seek veterinary attention immediately if the bandage becomes wet, slips, or causes distress.

Casts

- Bending of the bandage during weight-bearing can lead to pressure sores and can be prevented using casts and splints. Robert Jones bandages can bend soon after placement so are only a temporary solution.
- Make and incorporate a transarticular splint (e.g. using 6–10 folded layers of VetCast) to immobilise a joint, using at least two encircling loops of non-distensible tape around the padding and splint, above and below the joint.
- Casts usually have a very thin layer of padding underneath and can be 'bivalved' (cut from top to bottom medially and laterally) and tightly taped together again, to enable wound care.
- The owner must have written instructions on cast care and the dangers of ignoring clinical signs: swelling of the toes, chewing at the cast, distress, pain or smell. They must check the cast twice daily.
- The cast must be checked weekly by a member of the veterinary team.
- Casts used to support fracture repair are usually not changed for 6 weeks but must still be checked.
- Casts placed on the limbs of growing animals should be used with extreme care if needed for longer than one week, as this risks causing irreversible growth deformity

4.3.7 WOUND MANAGEMENT

- Wound healing is promoted by a moist environment and reduced inflammation.
  - Assess the injury:
    - Take a history.
    - Check for other organ injuries.
    - Check for deep damage, e.g. crushing of muscle/bone in bite wounds. Radiography and ultrasound are invaluable – look for unexpected free gas and free fluid.
    - Probe penetrating injuries, especially over the chest and abdomen.
    - Surgically explore bite wounds – clip wide in case body cavities are involved.
  - Control infection:
    - Promote clean healthy tissue.
    - Lavage with large volumes (1–5 l) of sterile fluid to remove debris and bacteria. Use a 5/8 inch (16mm) 21 g needle attached by a drip line to a

fluid bag inside a pressure bag, and pump this up to generate a suitable flow rate.
  - Always wear a mask as bacteria can be aerosolised.
  - Antiseptics are not needed.
  - Surgically debride obviously necrotic tissue initially, and again if required (rarely needed).
  - Use wet-to-dry dressings for further debridement.
  - Immediate intravenous antimicrobials are appropriate.
- Manage the wound environment:
  - Select an appropriate method of closure, e.g. primary closure (using a drain if needed), delayed primary closure (2–5 days) or secondary closure (5 days), or second-intention healing, after open wound management
  - Use semi-occlusive dressings (e.g. Allevyn) plus hydrogel (e.g. Intrasite gel) for further surface debridement, as the bandage does not need to be changed as often as for wet-to-dry dressings, allowing more healing with less intervention (cost, stress)
  - Once healthy granulation tissue covers the wound, ongoing moist wound care is not needed. The wound can be left open to contract (protected from trauma and dirt if needed, i.e. licking, lying down) or can be closed surgically.
- General patient management
  - Nutrition: consider oesophagostomy-tube feeding.
  - Analgesia: tissue injury and open wounds can be tender.
  - NSAIDs and bandages reduce pain, opioids may also be needed in the acute phase.
  - Antimicrobials are used initially but are not a substitute for lavage and debridement. Stop antimicrobials once the wound is healthy and granulating.

- Wounds that are best closed:
  - Those that can be converted to a clean-contaminated wound by lavage and debridement.
  - Thoracic wounds.
  - Tissues that need normal function, e.g. near eyes, over joints.
  - Skin wounds over fractures are best closed within 3 days.
- Wounds that are best left open even if just for a few days:
  - Contaminated or dirty wounds where wound closure will probably result in abscess formation.
  - Suspected ongoing ischaemia/necrosis.
  - Distal limbs where there is not much available skin.
  - Be aware of deep injury, e.g. necrosis in traumatic wounds such as bite wounds.

! If in doubt about the degree of contamination or ongoing necrosis, a wound should be left open. It can be closed in a few days if the wound is healthy or can be left for longer if there are continuing doubts.

POST-OPERATIVE CARE

- Nursing: post-op patients require warmth, comfort, fluid therapy, and analgesia. Delayed recovery may be due to hypothermia, hypotension, electrolyte imbalance, pain and fear, or dehydration.
- Analgesia and anxiolytics: reduce stress and improves recovery. Opioids can affect gastrointestinal motility.
- Nutrition: early nutrition is essential; there is no indication to withhold food. Feeding should be encouraged as soon as possible after recovery, including after GIT surgery. Nausea post-anaesthetic/gastric surgery can be treated with prokinetics, antacid medication or central/peripheral antiemetics. At the end of major surgery, anorectic animals should have a feeding tube placed.

Postoperative complications

- Complications may be due to wound, patient, or surgical factors.
    - ***Seroma***: a relatively common complication, especially when skin has been undermined, or in loose-skinned animals around the body folds or the neck. Most do not require treatment and will resolve over several weeks. Needle drainage is discouraged, as the seroma will re-form and there is a risk of iatrogenic infection. Large seromas may require surgical drain placement.
    - ***Surgical wound dehiscence***: usually due to excessive wound tension. Other causes include infection, tight sutures, inadequate analgesia, excessive movement, or self-trauma. Most cases respond well to open wound management and healing by second intention. Re-suturing is only performed if the underlying cause of dehiscence can be overcome and if tissue is healthy, or if critical structures or implants are exposed
    - ***Infection***: multi-drug-resistant infections, including methicillin-resistant *Staphylococcus pseudointermedius* (MRSP), are occasionally encountered; their management is the same as for other infections. Use cytology and bacterial culture and sensitivity to choose antimicrobial therapy.
    - ***Haemorrhage*** is usually due to poor surgical technique and inadequate haemostasis. Animals with haemorrhage in an abdominal cavity, e.g. after neutering, may present with weakness, collapse, pallor, slow recovery, and tachycardia. Aspiration of blood via abdominocentesis is diagnostic. Following intravenous fluid therapy, emergency surgery will be required. Rarely, haemorrhage is due to clotting disorders; petechiae and bleeding from catheter/venepuncture sites may also be evident.
    - ***Peritonitis***: dehiscence of a gastrointestinal incision if it occurs, is usually within 72 hours of surgery and will lead to septic peritonitis. Inappetence and vomiting are early presenting signs, and abdominocentesis yields fluid containing degenerate neutrophils and intracytoplasmic bacteria. Following intravenous fluid therapy, emergency surgery is required.

4.4 ORTHOPAEDICS

4.4.1 *Scope of this section* 4.4.2 *Approach to the lame dog or cat* 4.4.3 *Diagnostic tests and imaging* 4.4.4 *Forelimb disease* 4.4.5 *Hind limb disease* 4.4.6 *Degenerative joint disease (DJD)* 4.4.7 *Managing the chronic orthopaedic patient* 4.4.8 *Emergency management of appendicular fractures*

INTRODUCTION

4.4.1 SCOPE OF THIS SECTION

- The following is designed to address the approach to an orthopaedic case, with a brief discussion of common conditions.
- It is not possible within the scope of this text to cover individual orthopaedic procedures. For an approach to spinal disease see 3.11.

HISTORY AND EXAMINATION

4.4.2 APPROACH TO THE LAME DOG OR CAT

- The consultation should involve:
  - Gait analysis.
  - History taking.
  - Orthopaedic examination, including observation at rest.
- ***Gait analysis***: it is easiest to evaluate dogs at walk and trot on a firm level surface. Cats can be given free range in a large secure room.
  - Forelimb lameness (4.4.4) will result in head-nodding or head up on the painful limb.
  - Hind-limb lameness (4.4.5) is suggested when the hock is higher, to reduce weight bearing; the leg may also be held away from the body. The pelvis may be rotated towards the sound side during the swing phase of movement, reducing joint excursion.
  - Bilateral forelimb disease can be difficult to detect. Sufferers tend to shift their weight backwards and have a hunched back, crouched hind-limb stance and a very upright neck position, especially on rough surfaces.
  - Bilateral hind-limb disease tends to cause a wobbling gait and a shift of weight forward.
- ***History***: note duration, acuteness of onset, evidence of trauma, progression and persistence of the lameness.
  - Inactivity stiffness suggests osteoarthritis, elbow dysplasia, or cruciate injuries.
- ***Orthopaedic examination***: it can be difficult to perform a full examination in some patients, especially cats and small dogs. Sedation may be necessary.
  - Examine limb position, symmetry of weight bearing and musculature.
  - Examine ability to stand and stay standing.
  - Examine head, neck, and tail carriage.
  - Firmly palpate the spinal column and manipulate the neck – but if cervical disease is suspected, do not perform this manipulation.
  - Forelimbs may be examined standing, sitting, or in lateral recumbency; hind limbs, standing, or in lateral recumbency. If cruciate disease is suspected perform a tibial thrust test (Figure 4.3a).
  - All joints should be assessed for range of movement (ROM) (Table 4.3), discomfort, crepitus, thickening, instability, or effusion.
  - Examine soft tissues for change in size (swelling or atrophy), warmth, and discomfort.
  - Examine paws, pads, claws, interdigital skin, phalangeal bones and joints, and metacarpo/tarsophalangeal joints, for wear pattern, scuffing, and pain
- Some dogs and cats will not be lame when presented for examination, as their lameness is intermittent and the excitement of the consultation may mask low-grade pain. If the presence or source of lameness cannot be identified then:
  - Hospitalise and repeat the examination later in the day.
  - Perform a full neurologic examination and rule out non-orthopaedic disease.

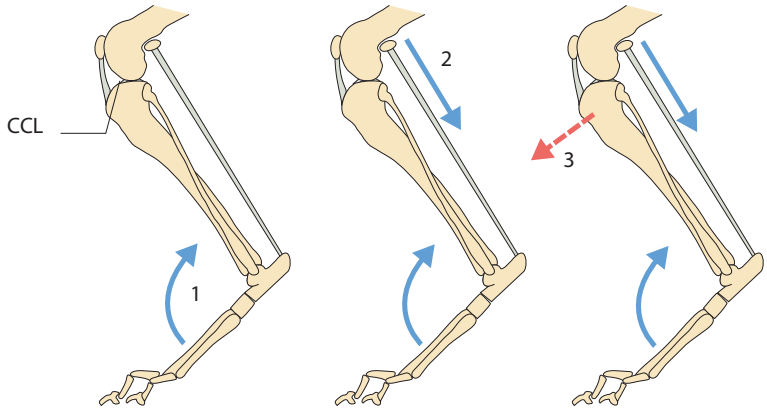


Figure 4.3a **Tibial thrust test**. The stifle is held in slight flexion. The index finger of one hand is placed over the tibial crest and the other hand flexes and extends the hock (1, 2). If the CCL is torn, the tibial tuberosity will move very slightly cranially (3), when the hock is in the flexed position.

Table 4.3 Normal Canine Joint Range of Movement

	Flexion	Extension
Shoulder (relative to scapular spine)	20–30°	155–160°
Elbow	15–20°	160–165°
Carpus	20–25°	190–210°
Hip (relative to axis of pelvis)	10–20°	170–180°
Stifle	15–25°	155–165°
Tarsus	15–25°	180–200°

- Ask the owner to obtain video evidence of lameness.
- Ask the owner to keep a 'lameness diary', and revisit on a 'bad' day.

DIAGNOSIS

4.4.3 DIAGNOSTIC TESTS AND IMAGING

- **Blood work** is generally unhelpful; it is most valuable in joint disease.
  - Consider routine haematology and C-reactive protein (for evidence of inflammation or infection), muscle enzymes, serology/PCR for Lyme disease; serology for Neospora or Toxoplasma.
  - Other infectious disease depending on travel history or local prevalence.
  - Anti-nuclear antibody (ANA) and rheumatoid factor (RF) are of limited value and not primary tests.
- ***Radiography*** is the most valuable initial investigation. Note that radiographic sensitivity is greatly improved if lameness has been localised.
  - Patient should be sedated or anaesthetised.
  - At least two views of each area are necessary, and preferably also the contralateral limb for comparison.
  - Stressed, flexed, and extended views may be helpful.
  - Good positioning and radiographic technique are essential.
    - Imaging of the soft tissues is as important as the bone.
  - Changes should be evaluated in conjunction with physical findings, age and breed.

- Joint fluid analysis: see 4.4.6.
- Ultrasound is increasingly used to examine soft-tissue structures supporting joints/muscles; interpretation requires experience.
- Advanced imaging: CT and MRI offer advantages over radiography but require specialist equipment and interpretation.
- Arthroscopy/arthrotomy are necessary to fully evaluate some cases with joint disease; they also allow therapeutic intervention. They require specialist equipment and knowledge to be performed well.
- Force plate with video analysis of gait measures the force as each limb is planted, to better evaluate lameness and response to treatment, while high-speed video cameras are used to analyse gait.
- Scintigraphy: technetium is used to look for focal 'hot spots' affecting the bone/support tissues, suggesting inflammation.

CAUSES OF LAMENESS

4.4.4 FORELIMB DISEASE

- Non-fracture, common causes of forelimb lameness include:
  - Carpal hyperextension injuries – torn ligaments as a result of a fall or trauma.
  - For immature dogs:
    - Elbow dysplasia (coronoid, osteochondritis dissecans [OCD], ununited anconeal process, radioulnar incongruity).
    - Antebrachial growth deformity such as carpal valgus.
    - Shoulder OCD in large and giant breeds.
- For adult dogs:
  - Elbow osteoarthritis (OA) – usually secondary to dysplasia.
  - Soft-tissue injury of the shoulder, such as ligament tear or tenosynovitis.

4.4.5 HIND-LIMB DISEASE

- Non-fracture, common causes of hind-limb lameness include the following:

Immature dogs

- Hip dysplasia.
- Medial patella luxation:
  - Grade I – manually displaces but spontaneously reduces.
  - Grade II – intermittently and spontaneously luxates, resulting in a skipping gait.
  - Grade III – permanently luxated but can be reduced.
  - Grade IV – permanently luxated and irreducible.
- OCD of the stifle and hock.

Adult dogs

- Cruciate ligament injury – usually cranial. Integrity of the cranial cruciate ligament (CCL) is evaluated using the tibial thrust test (Figure 4.3a).
- Hip OA.

COMMON CONDITIONS AFFECTING ANY LIMB INCLUDE THE FOLLOWING:

Immature dogs

- Metabolic disease – lameness and pain on palpation; may show systemic signs.
- Metaphyseal osteopathy.

Adult dogs

- OA: signs will wax and wane; worse after rest.
- Osteosarcoma, usually seen 'away from the elbow and near the stifles'.

JOINT DISEASE

4.4.6 DEGENERATIVE JOINT DISEASE

- Osteoarthritis associated with degenerative joint disease (DJD) is the most frequently occurring joint disease in dogs and cats. Diagnosis may require radiography or arthrocentesis (see below).

Differential diagnosis

- Traumatic OA usually affects a single joint with a history of trauma.
- Septic OA usually affects single joints.
- Polyarthritic (immune-mediated) OA:
    - Sufferers are usually systemically unwell, showing fever, leucocytosis, inappetence, and/or reluctance to exercise/lethargy – the phenomenon of 'walking on eggshells'.
    - Joint swelling and pain can be subtle.
    - Polyarthritic OA can be erosive or non-erosive:
    - Erosive polyarthritis is similar to rheumatoid arthritis in humans. In dogs, this is diagnosed from radiographs, as RF in dogs is insensitive and non-specific.
    - Non-erosive polyarthritis in its most common form is secondary to another underlying disease, and may be accompanied by pancreatitis, meningitis, etc. Infectious causes include tick-borne disease and chronic bacterial infection, e.g. prostatitis. Radiographs appear normal (with soft tissue swelling) and the condition is diagnosed via arthrocentesis.

Arthrocentesis procedure

- Arthrocentesis should be performed on 4–6 joints if polyarthritis is suspected (Figure 4.3b).
    - Use a 23 g needle for cats, and for tight joints in dogs (15–25 mm); use 21 g for larger joints in dogs (25–40 mm), except for the hip.
    - Releasing suction pressure prior to removing the needle reduces the likelihood of blood contamination.
    - If only a few drops are obtained, smear onto a slide; place larger amounts in EDTA.

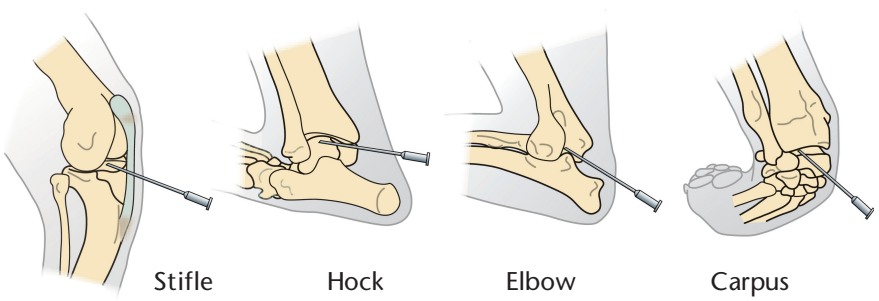


Figure 4.3b Anatomic approach to obtaining joint aspirates

- For culture, place in a sterile glass container and add nine times the volume of liquid culture medium (used for blood cultures).
- Table 4.4a outlines the interpretation of the joint fluid analysis.

TREATMENT

4.4.7 MANAGING THE CHRONIC ORTHOPAEDIC PATIENT

- The majority of chronic orthopaedic cases involve degenerative joint disease (DJD) leading to OA.
- A wide variety of therapies exists, so a coordinated treatment plan must be developed. Such plans are complicated by the lack of clear evidence of medical efficacy. The options include:
  - ***NSAIDs*** (4.2.4). It is important to realise that the disease process continues regardless of its expression as clinical signs, therefore chronic lower-dose therapy may be more effective.
  - ***Other pain relief*** includes opioids, bedinvetmab, frunevetmab, paracetamol (not cats), gabapentin, benzodiazepines, methocarbamol, amantadine/memantine, and amitriptyline (4.2.6).
  - ***Glycosaminoglycans*** (pentosan polysulphate) have been shown to have benefit in reducing pain.
  - ***Surgery***: the joint environment can be improved via:
    - Arthroscopy/arthrotomy procedures.
    - Replacement (hip or elbow).
    - Surgical arthrodesis.
  - ***Nutraceuticals***: a variety of products is available, either as nutritional supplements or incorporated in special diets. They include antioxidants (PUFA), glucosamine, chondroitin, green-lipped mussel, lysine, carnitine, methionine, turmeric and *Boswellia* extract.
  - ***Weight loss*** for patients with BCS of 5/9 or above.
  - ***Hydrotherapy***: underwater treadmill and supported swimming are available, and can help reduce bodyweight without over-straining the painful region
  - ***Physiotherapy***: several modalities have been shown to reduce pain, control inflammation and improve balance and ROM. They include cryotherapy, passive ROM exercises, stretching exercises, balance and proprioceptive exercises, massage therapy, therapeutic ultrasound, laser, transcutaneous electrical nerve stimulation (TENS), shock wave therapy, and active exercise.

Table 4.4a Effect of Disease on Synovial Fluid Analysis

	Volume (ml)	Viscosity	NCC* (x10^9/l)	Mononuclear (%)	Neutrophils (%)
Normal	0.1–1.0	High	<3	≥90	≤10
DJD or trauma	N–↑	↓	<5	≥90	≤10
Septic	↑↑	↓↓	>3	≤75	≥25
Immune-mediated	↑↑	↓↓	>3	≤85	≥20

* Nucleated cell count

  - Altering the home environment can be helpful, especially slippery flooring and stairs.
  - A home-exercise programme for the owner to manage, with 'dos and don'ts' that can be simply followed to keep joints moving, encouraging fun but low-impact activities such as retrieval from a water course
- Other modalities of therapy include acupuncture, homoeopathy, and magnets. The benefits of these are unproven.

4.4.8 EMERGENCY MANAGEMENT OF APPENDICULAR FRACTURES

! Fractures are rarely life threatening, but shock, pulmonary contusion, or rupture of the diaphragm/bladder can be. Ensure that these are covered by the initial patient assessment.

- Stabilise the patient before radiographing the fractures.
- Provide analgesia: methadone/morphine plus NSAID (if the patient is not hypotensive).
- If pain is non-responsive, consider neurologic injury, e.g. sciatic nerve entrapment.
- ***Open fractures***
  - Open fractures are classified according to their severity (Table 4.4b); management will depend on the grade of the fracture.
  - Cover with sterile dressing and change daily until fixation.
  - Administer broad spectrum IV antimicrobials – potentiated amoxicillin, cephalosporin – as the wound will be susceptible to hospital-acquired infection.
  - Once the patient is stable, use the following procedure for wound cleaning and lavage.
    - Give sedation or general anaesthetic.
    - Preparation: wear sterile gloves, disinfect clipper blades, pack wound with sterile KY jelly, clean skin with chlorhexidine.
    - Technique: use a 500 ml bag of warm sterile Hartmann's solution; connect the bag via a giving set to a three-way tap with a syringe and a 19 g needle attached.
    - Debride wound.
- ***Temporary stabilisation*** is achieved differently for distal and proximal fractures.
  - Distal limb fracture (below elbow/stifle) requires support dressing (a splint or Robert Jones bandages) that minimises further tissue damage and provides pain relief. Extend the dressing above the elbow/stifle.
  - Proximal limb, pelvic, or spinal fracture requires strict cage rest, with an effective spica splint, Schroeder–Thomas splint, or spinal board, if possible.

Table 4.4b Classification of Open Fractures

I	Wound <10 mm; trauma from inside–out; minor tissue damage
II	Wound >10 mm; trauma from outside–in; moderate tissue damage
IIIa	Extensive tissue damage; possible bone loss; reconstruction likely
IIIb	Extensive tissue damage; possible bone loss; reconstruction unlikely
IIIc	Extensive tissue damage; possible bone loss; major arterial injury

- ***Assessment UGA***
  - Radiograph the fracture site.
  - All limbs, including the rest of the injured limb, should be assessed, including joints to evaluate instability, crepitus, or luxation.
  - Other areas may also need assessment, e.g. thorax, urinary tract.

! One in eight pelvic fractures compromise the integrity of the urinary system.

  - Radiograph the whole bone with at least two orthogonal views, and assess for joint involvement, non-displaced fissure fractures and pre-existing pathology, e.g. lytic lesion.
  - Radiography of contralateral bone helps planning for fixation.

5

CRITICAL CARE

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5.1 FLUID THERAPY

- Fluid therapy (Tables 5.1a and 5.1b) replaces volume and can provide colloidal osmotic pressure (COP), as well as other benefits such as oxygen-carrying capacity.
- Correct choice maximises these benefits; the tables attempt to summarise characteristics which affect choice.
    - Ion concentrations: fluid choice should take account of the patient's blood biochemistry, e.g. if potassium is high, then Hartmann's or Haemaccel is not first choice because they will increase potassium still further.
    - Duration of action must be balanced against the maximum dose which can be given.
    - Base crystalloid rate on body surface area and age, but:
        - For immediate therapy 2 ml/kg/hr is a maintenance rate for adults and 4 ml/kg/hr for dogs/cats less than 4 months old. For hypovolaemic shock, give 60–90 ml/kg/hr (dog), 40–70 ml/kg/hr (cat).

5.2 HAEMORRHAGE: EMERGENCY MANAGEMENT

5.2.1 *Location and cause of bleeding* 5.2.2 *Controlling the bleeding* 5.2.3 *Emergency transfusion*

5.2.1 LOCATION AND CAUSE OF BLEEDING

- Treatment should be based on location and likely cause.

Table 5.1a Fluid therapy – composition of commonly used fluids

Crystalloids	**Na^+**	**K^+**	**Cl^-**	**Ca^{2+}**	**HCO_3^-**
7.2% NaCl	1232	0	1232	0	0
	Rapid volume expansion lasts 30–60 min 4–7ml/kg (dog), 2–4 ml/kg (cat)				
0.9% NaCl	150	0	150	0	0
	General use, especially low Na^+, high K^+ or Ca^{2+} or alkalotic patients				
Hartmann's	131	5	111	2	29
	Standard maintenance				
0.18% NaCl	30	0	30	0	0
	Hyperosmolar states, high Na^+				
5% dextrose	0	0	0	0	0
	Hyperosmolar states, drug diluents, never as a bolus				
Colloids	**Na^+**	**K^+**	**Cl^-**	**MW**	**COP**
Haemaccel*	145	5	145	30	25–28
Gelafusine*	77	0	62.5	35	33
Hydroxyethyl starch e.g. pentastarch have been largely withdrawn from human use due to the lack of benefit and increased risk of AKI. Use in veterinary patients has decreased although risk of AKI is unclear. Colloids generally are less used principal indications are for treatment of low blood volume or acute blood loss when crystalloids alone are insufficient. Potentially where adequate blood pressure cannot be maintained by crystalloids and pressor agents.					

MW = average molecular weight (kD) Ion concentrations are given in mmol/l

Table 5.1b Types of blood product	
Whole blood or packed red blood cells	Management of anaemia, essential group matched in cats, subsequent transfusions cross-matched. Start at 1 ml/kg/hr for 30 minutes and monitor patient for adverse response, then give remainder of bag over 3–4 hrs. Fresh blood also contains plasma proteins, clotting factors (CF) and platelets
Plasma	Fresh frozen (contains labile CF) or frozen, 10 ml/kg given as for blood; not volume replacing
Cryoprecipitate	Thawed centrifuged plasma, precipitate – labile CF, supernatant – non-labile CF, albumin and immunoglobulin
Albumin	Also provides COP. Human albumin can cause severe reactions (not seen in UK). 4 ml/kg of 20% solution raises albumin by 2.5 g/l
Xeno-transfusion	In the UK and other areas, cat blood is not readily available requiring a donor cat to be sourced for each transfusion. In an emergency, where the patient is likely to die before cat blood can be obtained, dog blood can be used, ideally fresh whole blood. A strong immune response will occur with survival at one week of cats receiving dog blood of 40%
Canine platelet concentrate	Variable availability, used in management of thrombocytopenia where there is or likely soon to be active bleeding. Limited evidence on survival impact

- Tranexamic acid acts to prevent fibrinolysis but seems to have a beneficial effect on patients that are haemorrhaging. 10–15 mg/kg slow IV over 15–20 minutes q8–12 hr (dogs and cats); 15–20 mg/kg PO q8 hr (dogs).
    - Common causes include trauma, neoplasia, thrombocytopaenia, and inflammatory conditions (haemorrhagic gastroenteritis, cystitis, pericarditis, and nasal disease).
    - Less common causes include angiostrongylus, hypertension, and amyloidosis.
    - ***History*** should focus on:
        - Likelihood of trauma, e.g. RTA, falls. Is the bleeding proportionate to any such trauma?
        - Previous responses following challenge to clotting, e.g. post-neutering.
        - Exposure to toxins (see xxx), especially coumarins.
    - If the bleeding point is not obvious:
        - Inspect carefully for ecchymoses or petechiation.
        - Perform urinalysis (dipstick plus sediment exam if dipstick is positive).
        - Image third spaces (pleural, abdominal, pericardial).
        - Image GIT: could there be GI bleeding?
        - Check: is this disseminated intravascular coagulation (DIC – a complication of many severe disease processes and of trauma)?

5.2.2 CONTROLLING THE BLEEDING

- Once the bleeding point *is* obvious:
    - Do a packed cell volume (PCV) and platelet count – manual smear examination.
    - Image the area of bleeding.
    - Assess the clotting (Table 5.2).
- If ***clotting is normal*** but there is vascular trauma:
    - Is the bleeding likely to stop without intervention?
    - Is local pressure going to be sufficient?
    - Is surgery appropriate? (see 4.3.3).

Table 5.2 Clotting tests

Parameter	Assessment
Buccal mucosal bleeding time (BMBT)	Global assessment of ability to form primary plug; abnormality implies vascular, platelet or von Willebrand factor defect/deficiency
Activated clotting time (ACT)	Extrinsic and common pathway
Activated partial thromboplastin time (APTT)	Factors XI, X, IX, VIII, and prothrombin deficiency Severe liver disease, DIC
Prothrombin time (PT)	Intrinsic and common pathway Factor VII (coumarin poisoning) and prothrombin deficiency Severe liver disease, DIC
Thromboelastometry and Thromboelastography	Evaluates platelet and clotting factor function – speed, size and quality of clot as well as duration (rate of fibrinolysis)

- If clotting is abnormal:
  - Is general supportive therapy going to be sufficient?
  - Will a transfusion be necessary? (5.2.3).

5.2.3 EMERGENCY TRANSFUSION

- If clotting is defective, is it due to platelet or clotting factor deficiency (5.2.2)?
  - For platelet deficiency, give fresh, whole blood or canine platelet concentrate (if available).
  - Platelets survive for <24 hr post transfusion.
- For clotting factor deficiency, give plasma, vitamin K (5.4.3).

5.3 TRAUMA AND RTA

- ***History***: a detailed history of the incident should include:
  - How long ago?
  - Is the patient improving, stable or deteriorating?
  - Other medical problems, or current medication, e.g. NSAIDs.
- ***Triage***: conduct a brief physical examination to assess:
  - Level of consciousness (LOC).
  - Respiratory rate and effort (5.5).
  - Heart rate and rhythm.
  - Perfusion: mucosal colour, pulse.
  - Temperature.
- ***Personnel needed***: ideally three (leader, monitor, 'gofer').
- ***Life-threatening conditions***: check 'Airway, Breathing, and Circulation' (ABC).
- ***Monitor***: begin a monitoring chart to evaluate trends:
  - Temperature, pulse, respiration.
  - Blood pressure and pulse oximetry.
  - Urine output.
  - PCV, total protein (TP), electrolytes, urea, glucose, blood gas.
- Establish IV access as soon as possible.
- If LOC is declining: consider metabolic, hypoxia, hypotension, toxins, drugs, primary brain pathology.
  - If patient also hyperexcitable, give sedative drugs, e.g. diazepam (5.8).
  - Progression: alert → depressed → stupor → coma.

  - The following actions should be considered:
    - Raise head and neck by up to 20°.
    - Secure airway against aspiration.
    - Try to maintain end-tidal CO_2 @ 30–35 mmHg.
    - O_2 therapy: maintain saturation >99%; pO_2 > 60%.
    - Avoid aggressive fluid therapy if possible.
    - If raised intracranial pressure is suspected, mannitol 0.25–1 g/kg can be given as a bolus over 20 minutes. Glucocorticoids are of no proven benefit.
- ***Heart rate and rhythm***: interpretation and response.
  - Tachycardia: usually extracardiac, e.g. blood loss.
  - Bradycardia: pressor, e.g. dobutamine 5–10 µg/kg/min CRI or ephedrine 0.05–0.2 mg/kg IV.
  - Arrhythmia: only intervene if other parameters suggest it is having a significant effect on cardiac output – most arrhythmia are better untreated.
- Response to poor perfusion:
  - If pale, hypotensive, normal PCV – use fluid resuscitation:
    - Crystalloid up to 90 ml/kg (dog), 70 ml/kg (cat) in the first hour.
    - If fluid ineffective then pressor agents.
  - If PCV significantly low – administer blood.
- ***Pain relief*** is an important consideration see 4.2

5.4 POISONING

5.4.1 *Poisons and common causes of poisoning* 5.4.2 *History, physical examination, and diagnosis* 5.4.3 *Specific antidotes: vitamin K, misoprostol* 5.4.4 *General therapy for a known poison* 5.4.5 *General therapy for suspected poisonings*

5.4.1 POISONS AND COMMON CAUSES OF POISONING

- Poisonings are not common in dogs and cats. In general, dogs tend to ingest poisons while scavenging, whereas cats ingest them while grooming.
- All pharmaceutical drugs, herbal products, and nutraceuticals are potentially poisonous. See 3.13.8 for antimicrobial side effects, 4.2 for analgesics and individual body systems in
- Section 3. Resources such as the Veterinary Poisons Information Service (VPIS UK]) can offer online (https://www.vpisglobal.com) and telephone help. For poisonous plants in cats, e.g. lilies – https://www.cats.org.uk/help-and-advice/home-and-environment/dangerous-plants

5.4.2 HISTORY, PHYSICAL EXAMINATION, AND DIAGNOSIS

- ***History*** is the key to appropriate therapy. Most poisonings have a clear history of known or likely exposure.
- Poisonings in cats and dogs are generally acute. Cases can present with a variety of clinical signs and are therefore included in many differential lists.
- ***Diagnosis***: while specific diagnostic tests are available for some poisons, results are slow. Such tests are therefore confirmatory rather than therapeutically useful.
  - Routine haematology, coagulation profiles, and biochemistry are helpful in establishing the specific poison, and how significantly affected the patient is.

5.4.3 SPECIFIC ANTIDOTES: VITAMIN K, MISOPROSTOL

- Few poisons have specific antidotes.
- Coumarins in anticoagulant rodenticides (ACRs) work by blocking production of vitamin K-dependent clotting factors.
- ***Initial vitamin K therapy***: Use a small bore (23g) needle, with the vitamin K volume divided at several sites to which pressure can be applied if bleeding occurs.
    - For a known warfarin, first-generation coumarin, indandione, or an unknown coumarin: vitamin K 2.5 mg/kg SC q8 hr.
    - For a known second-generation coumarin, such as brodifacoum: vitamin K 5 mg/kg SC q8 hr.
- ***Misoprostol*** is a prostaglandin analogue, although not specifically an antidote, can be used in known NSAID overdose or toxicity to reduce risk of gastric ulceration. NB abortifacient. Dogs – 2–5 µg/kg PO q8 hr; cats – 5 µg/kg PO q8 hr.

5.4.4 GENERAL THERAPY FOR A KNOWN POISON

- When the patient has been seen to contact/ingest toxin, or there is accidental overdose, the first action should be to prevent further absorption:
    - ***Wash*** skin with copious water. If using detergent, check that this will not increase the rate of absorption.
    - ***Emetics***: administer within 2 hours of ingestion, provided that the poison is not a hydrocarbon or a caustic. Note that feeding the patient a small, moist meal improves chances of adequate emesis. Emetics include:
        - Apomorphine can cause intense vomiting: 0.2 mg/kg SC or 20–40 µg. kg IV (dog only).
        - Washing soda crystal at back of tongue.
        - Salt (2 tsp) or mustard in cup of warm water.
        - Syrup of ipecacuanha 1:1 with water: 2–5 ml (cat) 10–20 ml (dog).
        - Xylazine will cause sedation: 3 mg (cat); 1–3 mg (dog) IM.
        - 3% hydrogen peroxide: 2 ml/kg PO.
- ***Caustic/corrosive toxins***: use milk with demulcents via naso-oesophageal or orogastric tube.
- ***Non-caustic/corrosive toxins:***
    - Gastric lavage with warm water/saline, under general anaesthesia.
    - Instil activated charcoal (1–3 g/kg), kaolin, chalk, or barium.
    - Some toxins, e.g. theobromine undergoes enterohepatic recycling so repeated administrations required.
    - Cathartics increase elimination of activated charcoal.
- Once absorption has been halted, the patient should be given symptomatic and supportive care appropriate to the toxic substance.

5.4.5 GENERAL THERAPY FOR SUSPECTED POISONINGS

- Ensure patient's airway and adequate ventilation.
- Support cardiac output.
- Monitor heart rate and rhythm, and blood pressure.
- Check PCV, renal, and hepatic parameters, electrolytes glucose, and acid/base status.
- Manage hydration using intravenous fluid therapy (IVFT) with crystalloids (Hartmann's is usually appropriate).
- Treat central nervous system (CNS) signs:

  - Manage seizures with benzodiazepines or barbiturates.
  - Manage excitation as for seizures, or with sedation (medetomidine, acepromazine (ACP)/opioid).
- Maintain body temperature within normal range.
- Once the patient is stable, establish diagnosis and treat appropriately.
- See Table 5.4 for clinical signs and management of common poisonings.

5.5 DYSPNOEA: EMERGENCY MANAGEMENT

EMERGENCY MANAGEMENT OF DYSPNOEA (FIGURE 5.5)

- For a clinical approach to dyspnoea see 2.3.
- The key is to determine the level at which oxygenation is failing and then target treatment to that area:
  - Obstruction of airflow, e.g. tracheal collapse, feline asthma.
  - Failure of diffusion across the alveolar membrane, e.g. pneumonia, pulmonary oedema.
  - Failure of lung circulation, e.g. cardiac disease, pulmonary hypertension, pulmonary thromboembolism, anaemia.
- Random therapy, e.g. giving furosemide to cases that do not have pulmonary oedema, can be counterproductive.
- Dyspnoeic patients, especially cats, are very unstable and will die if stressed too far. Sedation may be required to stabilise or carry out diagnostic procedures (see 4.1.2).
- Patients with significant pulmonary parenchymal disease carry a poor prognosis.

5.6 COLLAPSE: EMERGENCY MANAGEMENT

EMERGENCY MANAGEMENT OF COLLAPSE (FIGURE 5.6)

- Collapse can be associated with the terminal stages of many diseases due to malnutrition, dehydration, cachexia, and metabolic disturbances.
- Work from presenting signs to diagnosis, then treat the underlying disease.
- In the majority of cases the following are rational initial steps while waiting for results of imaging and minimum database:
  - Support adequate BP.
  - Conservative rates of crystalloids.
  - Supplemental oxygen.
  - Monitoring of temperature, pulse, respiratory and heart rate.

Table 5.4 Common poisonings

Poisoning	Clinical sign																					Management
	Abdominal pain	Anaemia/bruising	Blindness	Coma/collapse	Constipation	Convulsions	Depression/lethargy	Diarrhoea	Miosis/mydriasis	Dyspnoea	Excitement	Fever	Haematuria	Inappetence	Incoordination/ataxia	Jaundice	Muscle tremor	Oliguria/anuria	Paralysis	Salivation	Thirst	Vomiting	
Acids/alkalis	•							•												•		•	2, 3
Acorns	•	•			•		•	•						•				•				•	1, 2, 3, 5
Adder bites		•		•			•	•				•										•	2, 4
Alcohol				•		•	•				•				•								2
Ant powders, etc.				•		•		•	•	•		•										•	2
ACRs								•		•			•				•					•	2, 4
Batteries*																							2, 3, 5
Cannabis						•	•		•		•				•		•						1, 2, 3, 5
Carbon monoxide					•			•		•					•					•			2
Chocolate						•		•		•											•	•	2, 3
Christmas trees								•														•	2
Conkers	•							•											•			•	1, 2, 5, 6
Ethylene glycol			•	•		•	•							•		•					•		2, 4

Flower bulbs						•	•	•								•					•	2, 3
Grapes/raisins								•									•				•	1, 2, 3, 5
Lilies†							•							•				•		•	•	1, 2, 3
'Glow sticks'	•						•												•		•	2, 3
NSAIDs		•					•														•	1, 2, 4, 5
Permethrin						•	•			•						•						wash, 2
Slug pellets				•		•				•	•				•		•		•			2
Toad toxicity				•			•		•	•					•		•			•	•	flush mouth, 2
Xylitol (diet sugar)				•		•									•				•		•	2, 4

* Signs depend on the type of battery, location, and whether leaking
† *Lilium* and *Hemerocallis* spp. (cats)
1 = Emesis
2 = Supportive
3 = Activated charcoal and/or demulcent
4 = Specific therapy
5 = Removal surgically, endoscopically, or by lavage
6 = Laxative

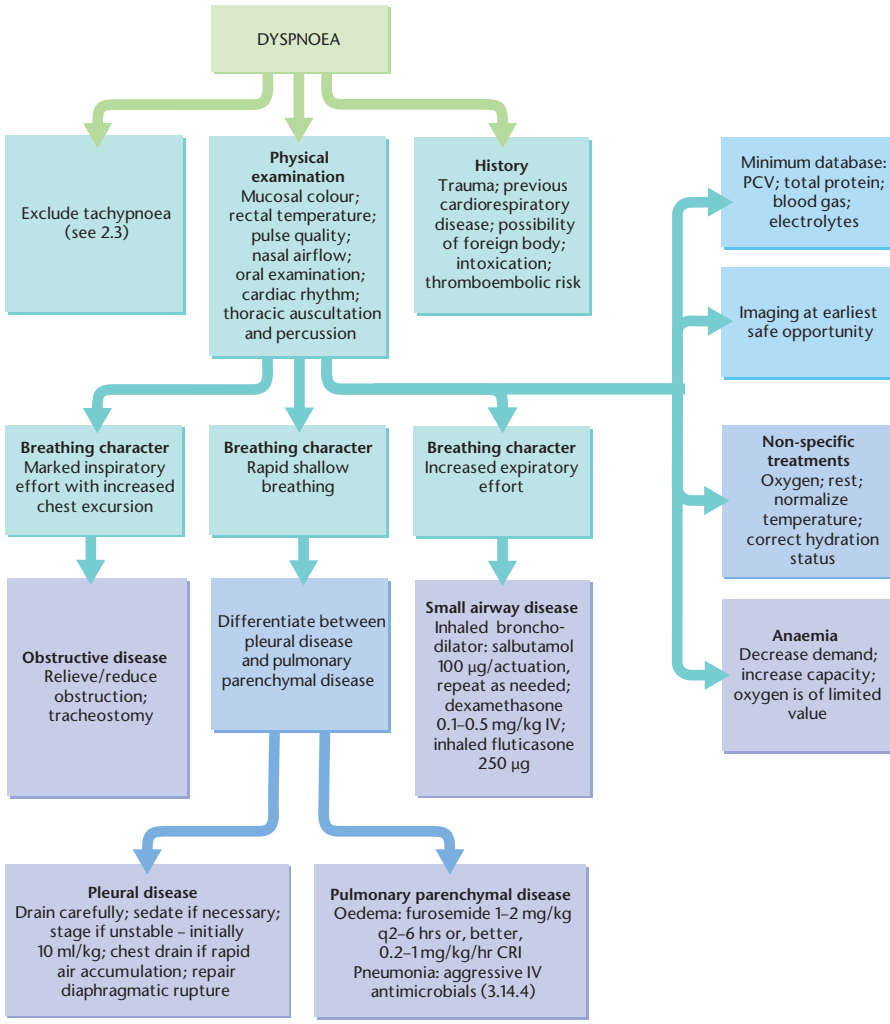
DYSPNOEA
Exclude tachypnoea (see 2.3)
Physical examination
Mucosal colour; rectal temperature; pulse quality; nasal airflow; oral examination; cardiac rhythm; thoracic auscultation and percussion
History
Trauma; previous cardiorespiratory disease; possibility of foreign body; intoxication; thromboembolic risk
Minimum database: PCV; total protein; blood gas; electrolytes
Imaging at earliest safe opportunity
Non-specific treatments
Oxygen; rest; normalize temperature; correct hydration status
Anaemia
Decrease demand; increase capacity; oxygen is of limited value
Breathing character
Marked inspiratory effort with increased chest excursion
Breathing character
Rapid shallow breathing
Breathing character
Increased expiratory effort
Obstructive disease
Relieve/reduce obstruction; tracheostomy
Differentiate between pleural disease and pulmonary parenchymal disease
Small airway disease
Inhaled broncho-dilator: salbutamol 100 µg/actuation, repeat as needed; dexamethasone 0.1–0.5 mg/kg IV; inhaled fluticasone 250 µg
Pleural disease
Drain carefully; sedate if necessary; stage if unstable – initially 10 ml/kg; chest drain if rapid air accumulation; repair diaphragmatic rupture
Pulmonary parenchymal disease
Oedema: furosemide 1–2 mg/kg q2–6 hrs or, better, 0.2–1 mg/kg/hr CRI
Pneumonia: aggressive IV antimicrobials (3.14.4)

Figure 5.5 Algorithm outlining the emergency approach to dyspnoea.

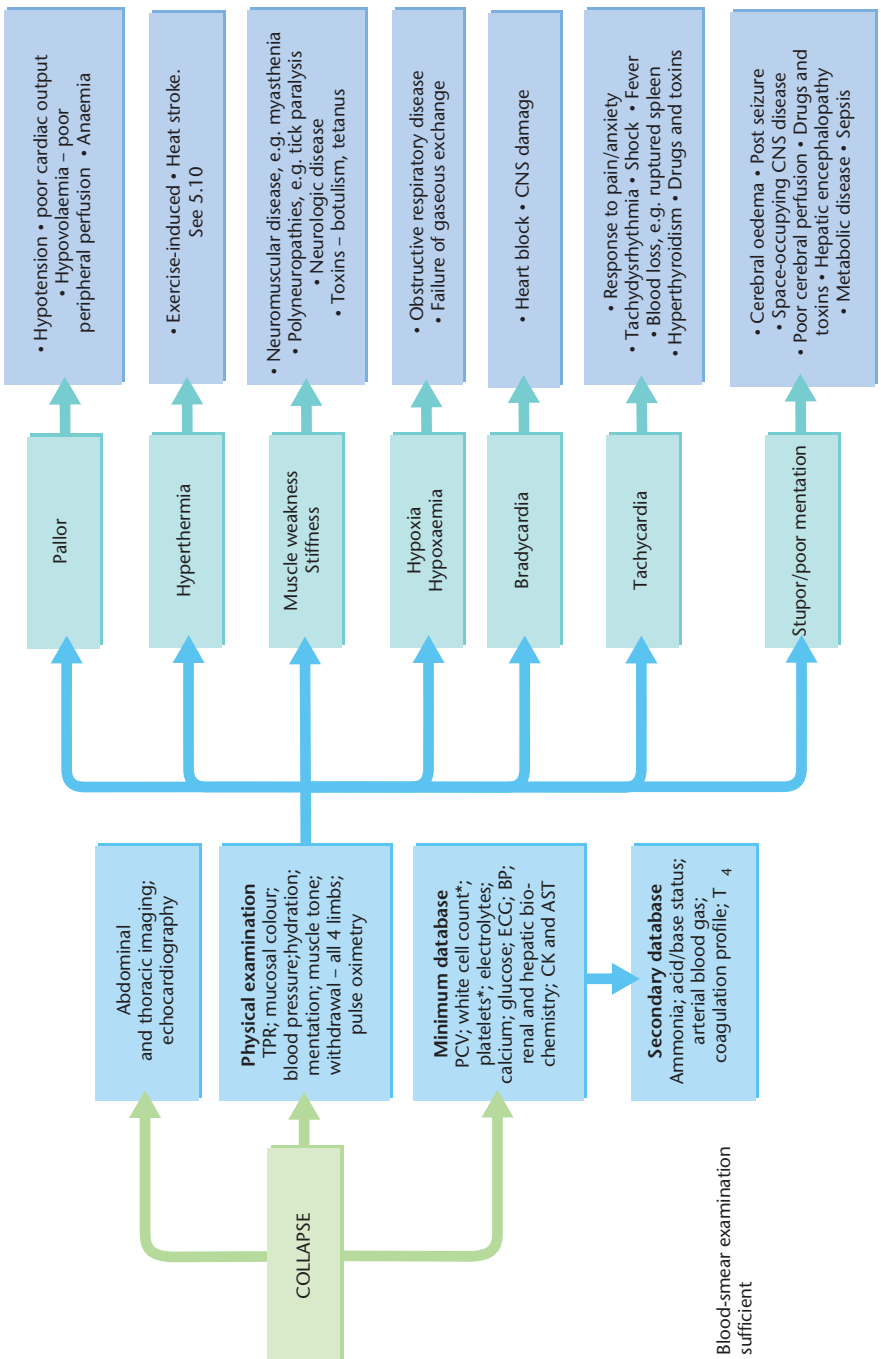
COLLAPSE
Abdominal and thoracic imaging; echocardiography
Physical examination TPR; mucosal colour; blood pressure;hydration; mentation; muscle tone; withdrawal – all 4 limbs; pulse oximetry
Minimum database PCV; white cell count*; platelets*; electrolytes; calcium; glucose; ECG; BP; renal and hepatic biochemistry; CK and AST
Secondary database Ammonia; acid/base status; arterial blood gas; coagulation profile; T4
* Blood-smear examination sufficient
Pallor
Hyperthermia
Muscle weakness Stiffness
Hypoxia Hypoxaemia
Bradycardia
Tachycardia
Stupor/poor mentation
• Hypotension • poor cardiac output • Hypovolaemia – poor peripheral perfusion • Anaemia
• Exercise-induced • Heat stroke. See 5.10
• Neuromuscular disease, e.g. myasthenia • Polyneuropathies, e.g. tick paralysis • Neurologic disease • Toxins – botulism, tetanus
• Obstructive respiratory disease • Failure of gaseous exchange
• Heart block • CNS damage
• Response to pain/anxiety • Tachydysrhythmia • Shock • Fever • Blood loss, e.g. ruptured spleen • Hyperthyroidism • Drugs and toxins
• Cerebral oedema • Post seizure • Space-occupying CNS disease • Poor cerebral perfusion • Drugs and toxins • Hepatic encephalopathy • Metabolic disease • Sepsis

Figure 5.6 Algorithm outlining the approach to the collapsed patient

5.7 URINARY OBSTRUCTION (FIGURE 5.7)

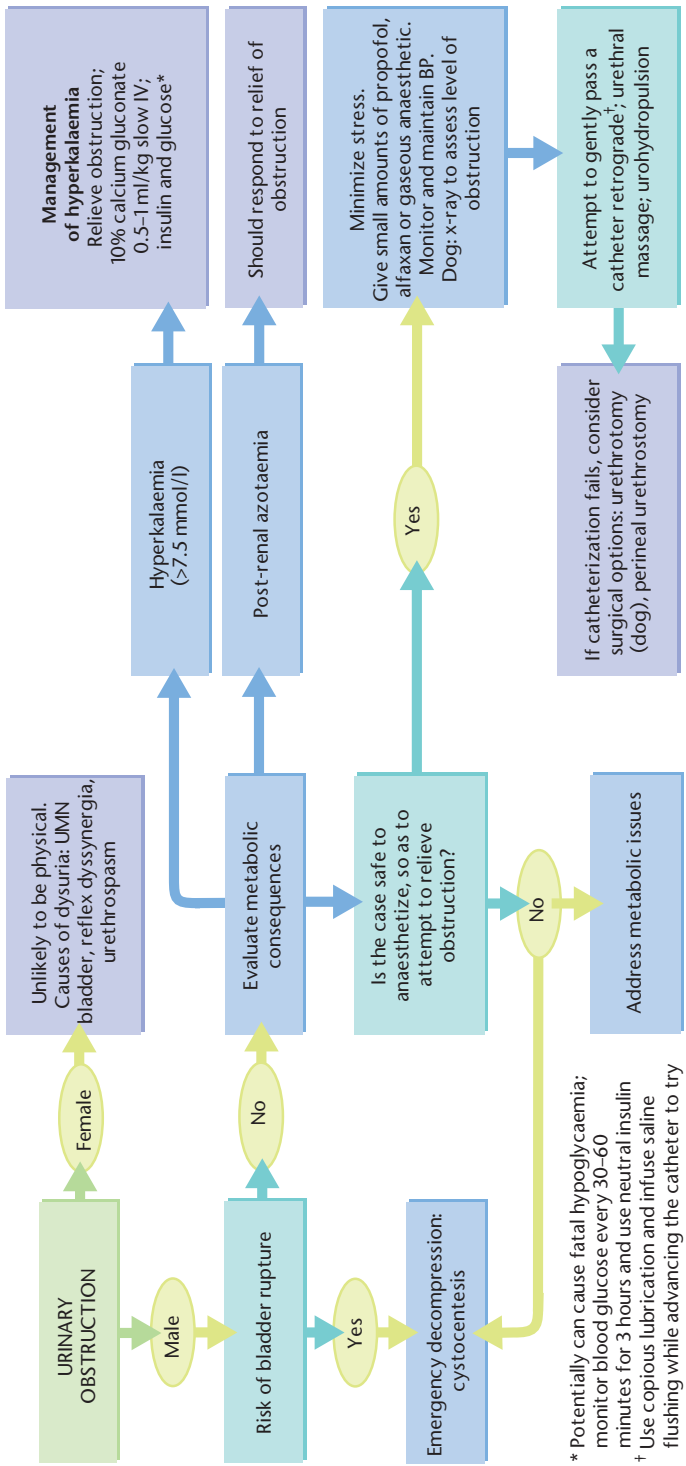
URINARY OBSTRUCTION
Male
Female
Unlikely to be physical. Causes of dysuria: UMN bladder, reflex dyssynergia, urethrospasm
Risk of bladder rupture
Yes
No
Emergency decompression: cystocentesis
Evaluate metabolic consequences
Hyperkalaemia (>7.5 mmol/l)
Post-renal azotaemia
Is the case safe to anaesthetize, so as to attempt to relieve obstruction?
Yes
No
Address metabolic issues
Management of hyperkalaemia
Relieve obstruction; 10% calcium gluconate 0.5–1 ml/kg slow IV; insulin and glucose*
Should respond to relief of obstruction
Minimize stress.
Give small amounts of propofol, alfaxan or gaseous anaesthetic.
Monitor and maintain BP.
Dog: x-ray to assess level of obstruction
Attempt to gently pass a catheter retrograde†; urethral massage; urohydropulsion
If catheterization fails, consider surgical options: urethrotomy (dog), perineal urethrostomy
* Potentially can cause fatal hypoglycaemia; monitor blood glucose every 30–60 minutes for 3 hours and use neutral insulin
† Use copious lubrication and infuse saline flushing while advancing the catheter to try and expand the urethra

Figure 5.7 Algorithm outlining the approach to urinary tract obstruction.

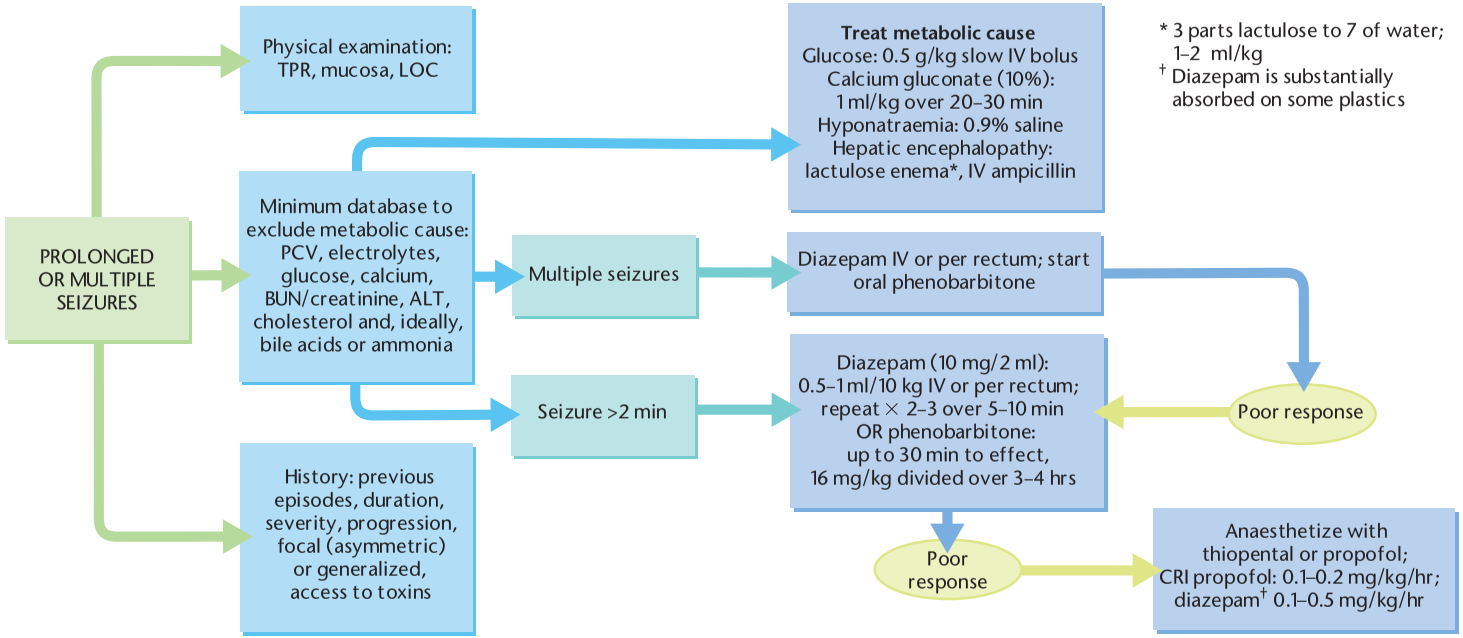
PROLONGED OR MULTIPLE SEIZURES
Physical examination: TPR, mucosa, LOC
Minimum database to exclude metabolic cause: PCV, electrolytes, glucose, calcium, BUN/creatinine, ALT, cholesterol and, ideally, bile acids or ammonia
History: previous episodes, duration, severity, progression, focal (asymmetric) or generalized, access to toxins
Treat metabolic cause
Glucose: 0.5 g/kg slow IV bolus
Calcium gluconate (10%): 1 ml/kg over 20–30 min
Hyponatraemia: 0.9% saline
Hepatic encephalopathy: lactulose enema*, IV ampicillin
Multiple seizures
Diazepam IV or per rectum; start oral phenobarbitone
Seizure >2 min
Diazepam (10 mg/2 ml): 0.5–1 ml/10 kg IV or per rectum; repeat × 2–3 over 5–10 min
OR phenobarbitone: up to 30 min to effect, 16 mg/kg divided over 3–4 hrs
Poor response
Poor response
Anaesthetize with thiopental or propofol; CRI propofol: 0.1–0.2 mg/kg/hr; diazepam† 0.1–0.5 mg/kg/hr
* 3 parts lactulose to 7 of water; 1–2 ml/kg
† Diazepam is substantially absorbed on some plastics

Figure 5.8 Algorithm outlining the emergency approach to the seizuring patient.

5.9 CARDIOPULMONARY CEREBRAL RESUSCITATION (FIGURE 5.9)

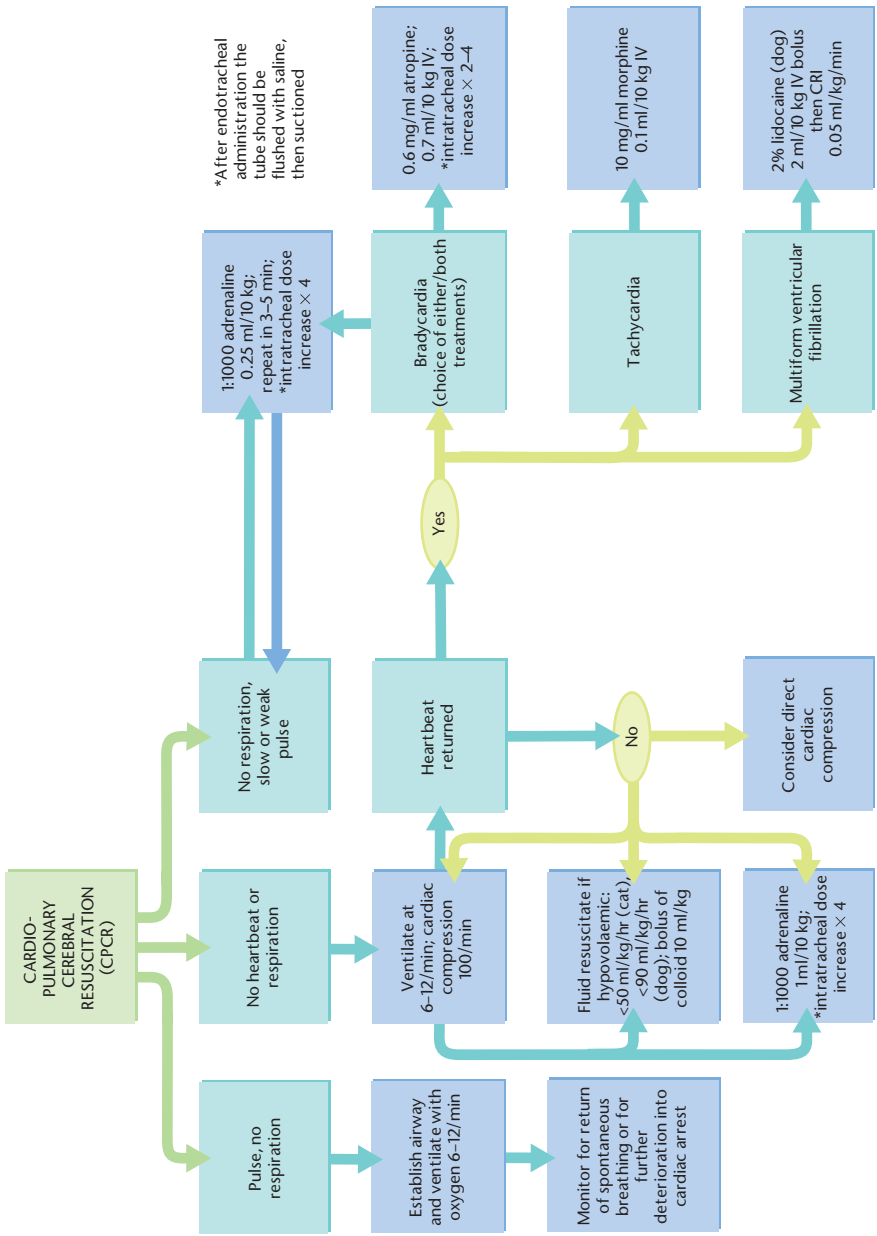
CARDIO-PULMONARY CEREBRAL RESUSCITATION (CPCR)
Pulse, no respiration
No heartbeat or respiration
No respiration, slow or weak pulse
Establish airway and ventilate with oxygen 6–12/min
Ventilate at 6–12/min; cardiac compression 100/min
Heartbeat returned
Monitor for return of spontaneous breathing or for further deterioration into cardiac arrest
Fluid resuscitate if hypovolaemic: <50 ml/kg/hr (cat), <90 ml/kg/hr (dog); bolus of colloid 10 ml/kg
No
Yes
1:1000 adrenaline 1ml/10 kg; *intratracheal dose increase × 4
Consider direct cardiac compression
1:1000 adrenaline 0.25 ml/10 kg; repeat in 3–5 min; *intratracheal dose increase × 4
*After endotracheal administration the tube should be flushed with saline, then suctioned
Bradycardia (choice of either/both treatments)
0.6 mg/ml atropine; 0.7 ml/10 kg IV; *intratracheal dose increase × 2–4
Tachycardia
10 mg/ml morphine 0.1 ml/10 kg IV
Multiform ventricular fibrillation
2% lidocaine (dog) 2 ml/10 kg IV bolus then CRI 0.05 ml/kg/min

Figure 5.9 Algorithm outlining basic CPCR.

APPROACH TO CPCR

- Personnel – ideally, four people are required to run resuscitation:
  - Person 1: ventilating the patient.
  - Person 2: performing cardiac compression.
  - Person 3: administering fluids and drugs.
  - Person 4: monitoring patient and keeping records of vital parameters, procedures and drugs given.
- Procedure requires you to:
  - Establish an airway using a cuffed endotracheal tube; if this is not possible, then perform tracheostomy to allow controlled ventilation.
  - Establish IV access.
  - Maximise monitoring: pulse/heart rate and rhythm, respiration, temperature, mucosal colour, PLR, level of consciousness, ECG, pulse oximetry, end-tidal CO_2, electrolytes, blood gas, and lactate.

5.10 HEAT STROKE (ACUTE HYPERTHERMIA) (FIGURE 5.10)

Most common in dogs left in cars on hot days, brachycephalic breeds at increased risk. Also, dogs excessively exercised on hot days especially if underlying disease, e.g. laryngeal paralysis; cats in tumble dryers.

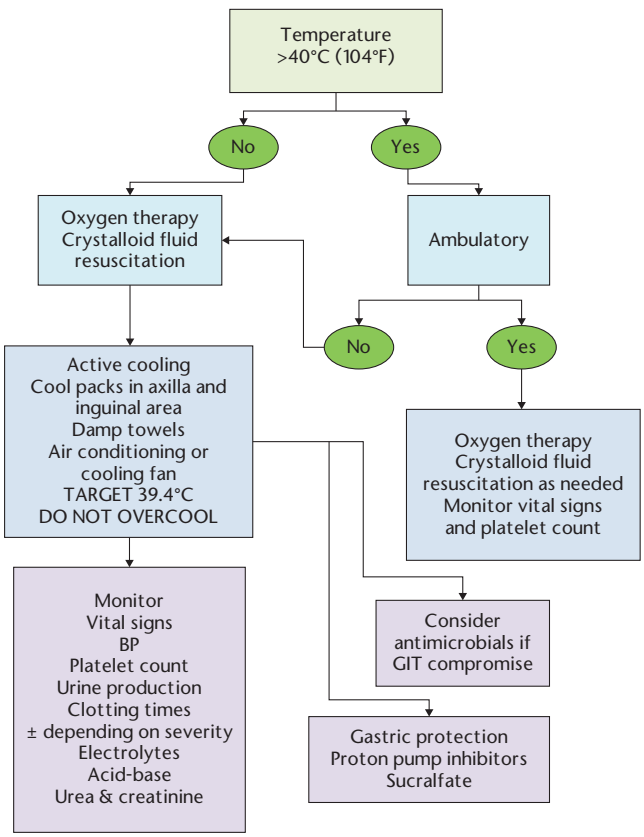

Figure 5.10 Algorithm outlining the approach to the patient with heat stroke.

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UNITS OF MEASUREMENT

Measurement	SI units	Traditional	Traditional—SI*	SI—traditional*
Serum biochemistry				
Albumin	g/l	g/dl	10	0.1
Bile acids	µmol/l	µg/ml	2.55	0.392
Bilirubin	µmol/l	mg/dl	17.1	0.058
Calcium	mmol/l	mg/dl	0.25	4
Chloride	mmol/l	mEq/l	1	1
Cholesterol	mmol/l	mg/dl	0.026	38.7
CO_2	mmol/l	mEq/l	1	1
Creatinine	µmol/l	mg/dl	88.4	0.011
Glucose	mmol/l	mg/dl	0.056	18
Phosphate	mmol/l	mg/dl	0.323	3.1
Potassium	mmol/l	mEq/dl	1	1
Protein	g/l	g/dl	10	0.1
Sodium	mmol/l	mEq/l	1	1
Urea nitrogen	mmol/l	mg/dl	0.357	2.8
Hormones				
ACTH	pmol/l	pg/ml	0.220	4.51
Aldosterone	pmol/l	ng/dl	27.7	0.036
Cortisol	nmol/l	µg/dl	27.59	0.036
Insulin	pmol/l	µU/ml	7.18	0.139
Oestrogen	pmol/l	pg/ml	3.67	0.273
Progesterone	nmol/l	ng/ml	3.18	0.315
Testosterone	nmol/l	ng/ml	3.47	0.288
Thyroxine (T_4)	nmol/l	µg/dl	12.87	0.078
Triiodothyronine (T_3)	nmol/l	µg/dl	0.015	64.9

* multiplication factors per unit

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SELECTED ABBREVIATIONS

ACEi	angiotensinconverting enzyme inhibitors
ACTH	adrenocorticotrophic hormone
ALKP	alkaline phosphatase
ALT	alanine amino-transferase
ARF	acute renal failure
BA	bile acids
BCS	body condition score
BSA	body surface area
CN	cranial nerve (CNI–CNXII)
COX	cyclooxygenase
CRD	chronic renal disease
CRI	continuous rate infusion
CT	computed tomography
DIC	disseminated intravascular coagulopathy
DM(B)	dry matter (basis)
ELISA	enzyme-linked immunosorbent assay
FCE	fibrocartilagenous embolus
FeLV	feline leukaemia virus
FGF	free gas flow
FIP	feline infectious peritonitis
FIV	feline immuno-deficiency virus
FLUTD	feline lower urinary tract disease
FNA(B)	fine needle aspirate (biopsy)
FORL	feline odontoclastic resorptive lesions
GFR	glomerular filtration rate
GI(T)	gastrointestinal (tract)
GME	granulomatous meningoencephalitis
HAC	hyperadrenocorticism
HCM	hypertrophic cardio-myopathy
H(L)	high (low)-dose
DDST	dexamethasone suppression test
IRIS	International Renal Interest Society
LMN	lower motor neurone
LOC	level of consciousness
MER	maintenance energy requirement
MRI	magnetic resonance imaging

NSAID	non-steroidal anti-inflammatory drug
NTI	non-thyroidal illness
NT-pro-BNP	N-terminal pro-brain natriuretic peptide
OA	osteoarthritis
OCD	osteochondritis dissecans
PCR	polymerase chain reaction
PDH	pituitary-dependent hyperadrenocorticism
PLi	pancreatic-specific lipase
PLR	pupillary light response
PP	polyphagia
PSS	portosystemic shunt
PTH (-RP)	parathyroid hormone (-related peptide)
PU/PD	polyuria/polydipsia
PUFA	polyunsaturated fatty acids
PUO	pyrexia of unknown origin
RER	resting energy requirement
ROM	range of movement
(T)T_4	(total) thyroxine
TLI	trypsin-like immunoreactivity
TSH	thyroid-stimulating hormone
(U)GA	(under) general anaesthetic
UMN	upper motor neurone
UPC	urine protein: creatinine ratio
UTI	urinary tract infection
VI	virus isolation

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